# Health and Well-Being in Adolescence

## Part one
## Physical Health and Subjective Well-Being

# Health and Well-Being in Adolescence

## Part one
## Physical Health and Subjective Well-Being

**Edited by Maria Kaczmarek**

**Bogucki Wydawnictwo Naukowe • Poznań 2011**

Supported by research funds 2008–2011 and co-financed by a grant from Iceland, Liechtenstein and Norway through the EEA Financial Mechanism and the Norwegian Financial Mechanism. Project ADOPOLNOR PL0255.



Reviewed by Napoleon Wolański



ISBN 978-83-62662-67-8

Bogucki Wydawnictwo Naukowe
ul. Górna Wilda 90, 61-576 Poznań
tel. +48 61 8336580
e-mail: bogucki@bogucki.com.pl
www.bogucki.com.pl

Printed in Poland
by Uni-Druk

# Contents

# Contributors


JOACHIM CIEŚLIK Department of Human Biological Development, Institute of Anthropology, Faculty of Biology, Adam Mickiewicz University, Poznań.

MAGDALENA DURDA Department of Human Biological Development, Institute of Anthropology, Faculty of Biology, Adam Mickiewicz University, Poznań.

JULIA DURZYŃSKA Department of Molecular Virology, Institute of Experimental Biology, Faculty of Biology, Adam Mickiewicz University, Poznań.

ANNA GOŹDZICKA-JÓZEFIAK Department of Molecular Virology, Institute of Experimental Biology, Faculty of Biology, Adam Mickiewicz University, Poznań.

TOMASZ HANĆ Department of Human Biological Development, Institute of Anthropology, Faculty of Biology, Adam Mickiewicz University, Poznań.

MARIA KACZMAREK Department of Human Biological Development, Institute of Anthropology, Faculty of Biology, Adam Mickiewicz University, Poznań.

ALICJA KRZYŻANIAK Department of Epidemiology, Chair of Social Medicine, Poznań University of Medical Sciences, Poznań.

MAŁGORZATA KRZYWIŃSKA-WIEWIOROWSKA Department of Epidemiology, Chair of Social Medicine, Poznań University of Medical Sciences, Poznań.

JANUSZ MACIASZEK Department Theory of Physical Education and Anthropomotorics, Faculty of Physical Education, Eugeniusz Piasecki University School of Physical Education, Poznań.

WIESŁAW OSIŃSKI Department Theory of Physical Education and Anthropomotorics, Faculty of Physical Education, Eugeniusz Piasecki University School of Physical Education, Poznań.

JOANNA PACHOLSKA-BOGALSKA Department of Animal Physiology and Development, Institute of Experimental Biology, Faculty of Biology, Adam Mickiewicz University, Poznań.

ALDONA SIWIŃSKA Department of Pediatric Cardiology and Nephrology, Poznań University of Medical Sciences, Poznań.

MAGDALENA SKRZYPCZAK Department of Human Biological Development, Institute of Anthropology, Faculty of Biology, Adam Mickiewicz University, Poznań.

BARBARA STAWIŃSKA-WITOSZYŃSKA Department of Epidemiology, Chair of Social Medicine, Poznań University of Medical Sciences, Poznań.

ROBERT SZEKLICKI Department Theory of Physical Education and Anthropomotorics, Faculty of Physical Education, Eugeniusz Piasecki University School of Physical Education, Poznań.

Maria Kaczmarek

# Why adolescence?

The term *adolescence* appeared into English in the fifteenth century as a derivative of a Latin word *adolescere*, which means to grow up or to grow into maturity [Muuss 1990]. The Random House Dictionary defines adolescence as "…the process or condition of growing up; the growing age of human beings; growing to manhood or womanhood; a transitional phase of growth and development between childhood and adulthood…". In the above meaning, adolescence is a transitional period, often defined as the period in life that serves as the bridge between childhood and adulthood.

In the classical world, Plato (c. 427–347 B.C.) and Aristotle (384–322 B.C.) initiated philosophical discussions of adolescence. They stated that lifespan is a process and it involves various stages. Aristotle proposed stages of life that might be included in contemporary models of youth development. He distinguished three successive, 7-year stages of life i.e. infancy, boyhood, and young manhood, prior to the person's attainment of full adult maturity, and described puberty as a distinct stage of life: "When twice seven years old in the most of cases the male begins to engender the seed, and at the same time hairs appear on the pubes. At the same time in the female the breast swell and the so called catamenia [e.g. menstrual discharges] commence to flow … in the majority of cases catamenia are noticed by the time the breasts have grown to the height of two finger breadths" [Tanner 1981:7].

However, in most of the time that elapsed between these initial philosophical discussions of adolescence and the present, this stage of life has not been distinguished in the human life history. Developmental changes occurring at the second decade of life were narrowly equated with puberty and reproductive maturity. A person usually moved from the status of child directly to the status of adult through socially recognized rites of passage, a cross-cultural phenomenon currently practiced in many native societies.

A concept of adolescence has developed since the late 18th century in its biological, psychological and social implications. Since that time and through the 19th century, biologists and physicians undertook more formal study of adolescent phenomena. Most research in the area concentrated on aspects of physical growth and sexual maturation during puberty, in particular the onset of menarche in females and seminal emission in males [Tanner 1981].

In the 1890s, psychologists began investigating the development, adjustment and behaviours of young people between the onset of puberty and marriage. All of

these endeavours ultimately led to the first complete definition of adolescence. It was given by an American psychologist, Granville Stanley Hall (1844–1924) in his two-volume work entitled *Adolescence: Its Psychology and Its Relations to Physiology, Anthropology, Sociology, Sex, Crime, Religion and Education* published in 1904 [Hall 1904]. Works of Hall and his followers provided adolescence as a formal concept and gave impetus and direction to the development of psychology, education, and adolescent culture.

As the changes in biological, psychological, and social development have become uncoupled, the study of adolescence has shifted. The history of the study has had two overlapping phases and is now on the emergence of a third one [Tanner 1999; Steinberg and Lerner 2004; Bogin 2005]. The first phase, which began in the early 20th century and lasted about 70 years, was characterized by descriptive study that purportedly related to all facets of adolescent development. The second phase, which began in the latter half of the 20th century and continues today, has examined stages of life cycle prior to adulthood in the course of human evolution and developmental plasticity. Such approach allowed pushing forward our understanding of changes in the adaptation of contemporary populations and promoting positive development of young people. We are now seeing the emergence of a third phase, in which the field of adolescent development becomes a noticeably interdisciplinary (a holistic) approach with a call to create a comprehensive view on adolescent stage of life. Researchers in various fields (human auxology, sociology, psychology, neurosciences and many others) are using the term adolescence to apply to the particular stage of life when distinct physical and psycho-social changes occur, thereby making it a formal biological, psychological, and even legal category.

Summarizing, the concept of adolescence is associated with an evolutionary process of human development which commences with puberty and lasts five to eight years, involving a rapid growth of height and weight (pubertal growth spurt), almost complete eruption of permanent teeth, development of secondary sex characteristics with the ultimate maturation of sexuality. Alongside with physical changes goes cognitive and intellectual, emotional and "…socio-sexual maturation, intensification of interest and practice in adult social, economic, and sexual activities" [Bogin 2005:55]. Thus, a capital is created for young people to enter their adult lives with.

A person in adolescence is called *adolescent* or *youth*. The terms *adolescents, young people* and *youth* are used interchangeably in relation to people aged 10–24 years. The UN and WHO use the term adolescents for people aged 10–19 years [UN 2008, WHO 2008]. The term young people, refers to those aged 10–24 years, and youth for those aged 15–24 years [UN 2008].

Current interest in the period of adolescence has been stimulated by recent social and demographic changes. Due to overall improvements of living conditions, improved nutrition, better hygiene, advances in control of diseases and other environmental modifications, the probability of dying during the first few years of life has substantially decreased. This resulted in the explosion of population growth. In 2009, there were 1.2 billion adolescents aged 10–19 in the world, representing 18% of the world population. Of them, 88% live in the developing and 12% in in-

dustrialized countries, the latter reflecting the sharp ageing of Europe and Japan. Around 50% of the world's adolescents live in urban areas. Migration trends from rural to urban areas will continue to intensify in the coming decades and by 2050 this proportion will rise to almost 70%, with the strongest increase occurring in developing countries. There is also a gap in gender parity, in favour of adolescent boys, in all regions of the world, the greatest in Asia and smallest in Africa.

Population of young people, aged 10–19 years, is now the largest in history, having more than doubled since 1950. This rising trend will continue in absolute terms until around 2030 [UNFPA 2006; UN 2009].

Over past 100 years, children have been growing and developing faster, becoming taller and maturing earlier, reaching physical and reproductive maturity at earlier ages, and achieving larger adult sizes than perhaps ever before in human history [Danker-Hopfe 1986; Bielicki and Szklarska 1999; Cole 2000; Olszewska and Łaska-Mierzejewska 2008; Wolański 2008].

Secular trends in the world are clearly linked to the changes related to environmental improvements, specifically changes in nutrition and health practices.

The acceleration in physical and reproductive maturity however, has been mismatched with social maturity. The time interval between attaining puberty and taking on adult roles (such as marriage and employment) has stretched out over past decades, from a 2 to 4-year to an 8 to15-year period, lengthening the transition from childhood to adulthood. Modern societal shifts in marital and reproductive patterns have expanded the gap between sexual maturation and marriage and between marriage and childbearing [Gluckman and Hanson 2006].

The widening age gap between biological maturity and psychosocial transition into adulthood is one of the possible explanations of a growing vulnerability for risk-taking behaviours such as dangerous driving, unplanned episodes of casual sex, and frequent turn-over of sexual partners, unsafe premarital sex with adolescent pregnancy and childbearing, and experimentation with alcohol, smoking cigarettes, drug abuse, poor dietary habits and physical inactivity [Lear 1995; Heaven 1996:76; DiClemente et al. 2009].

Chassin and Hussong [2009] highlighted some key characteristics of adolescence that enable us to understand the liability of young people to risky behaviours: increased sensitivity to immediate rewards, a focus on peers and social rewards, immature inhibitory and self-regulatory processes, increased risk taking and sensation seeking, and difficulty in mood regulation.

Adolescent emotional disturbances may lead to anxiety, depression, and dysfunctional relationships [Moore and Rosenthal 1992; Garber 2006]. Recent findings have shown that psychotic disorders rise steeply in early adolescence, and this is especially true for girls [Swarr and Richard 1996].

Young people may also run a high-risk of excessive impact of exposure to media on their decision making [Steinberg 2004, 2007; DiClemente et al. 2009].

Problems with control of behaviour and emotion may result in serious health problems, with some of them eventually leading to premature death. Young people may be killed or seriously injured in road accidents, commit suicides, experience bullying, and teen on teen violence, suffer from chronic diseases and disability, ex-

perience exclusion, marginalization, and loneliness. An alarming global health statistics indicate that approximately three-fourths of deaths occurring each year among persons aged 10–24 years are related to preventable causes such as motor-vehicle crashes, homicide, suicide, and other injuries (e.g., drowning, poisoning, and burns) [WHO 2008].

Unlike infants and children whose morbidity and mortality are mostly caused by infectious diseases, adolescents are featured by other conditions. These include diabetes, asthma, mental disorder, sexually transmitted infections such as HIV/AIDS, venereal diseases, herpes viruses/cancer diseases, epilepsy, and cancer [WHO 2008]. It is estimated that currently at least 12% of adolescents live with one chronic condition [Sawyer et al.2007; WHO 2008]. Recent studies have also shown the occurrence of chronic diseases in adolescence and their persistence into adulthood [Varraso et al. 2005]. It is estimated that approximately 70% of premature deaths among adults are caused by health-risk behaviours that begin during adolescence [WHO 2008]. These facts contradict the belief that adolescence is a period of life without a heavy "burden of disease" as compared to infancy and childhood. The health paradox of adolescence is that a peak in lifetime physical health is paradoxically accompanied by high mortality and morbidity. Indeed, adolescent physical maturation pushes an individual into peaks in physical growth, improvements in strength, speed, reaction time, and other capacities associated with lifespan and fitness [Tanner 1962; Metcalf and Monaghan 2003; Malina et al. 2004; Bogin 2005; Wolański 2006]. However, its effects on health and well being are profound and paradoxical as described above.

Although young people are generally viewed as a uniform group, they seem to respond to health-risk behaviours in diverse ways [King et al. 2009]. Therefore, significant variance in terms of the age of onset of substance use, the speed with which they escalate the behaviour, and their degree of persistence with certain patterns of use and abuse is observed [Chassin 2009]. Some evidence indicates that young people who begin to smoke at an earlier age are more likely than later starters to attempt suicide and engage in high-risk sexual behaviours [Chassin 2009].

As in the case of infants and children whose leading causes of morbidity can be prevented by immunization and the use of antibiotics (the communicable childhood diseases), or by improved sanitation (diarrheal diseases and gastroenteritis), some adolescent comorbidities (alcohol consumption and depression, uncontrolled sexual activity and STDs) should be modifiable [van der Veen 2001; Murray et al. 2005]. That is why, recent research is more directed toward adolescence taken as a time of opportunity to the adolescent positive health and development. To regard adolescence as a "gateway to health" is a relatively new notion. The approach known as positive youth development (PYD) is a comprehensive framework outlining the positive attributes young people need in order to become successfully contributing adults. PYD is both a philosophy and an approach to policies and programmes that involve and engage young people as equal partners. The underlying philosophy of youth development is holistic, preventative and positive, focusing on the development of assets and competencies in all young people [Hawkins and Weis 1985].

Specific health and development needs and challenges that hinder adolescents' well – being expose needs for strategies and special interventions aiming in improving adolescents' health across the globe and "…to foster a new generation of productive adults who can help their communities progress" have been recognized [WHO 2008]. According to the WHO declaration "Young people are our human capital for the future. Healthy people are not only more economically productive; they also make fewer demands on the health and welfare system. It will be the current generation of young people who will create the necessary economic activity to support the growing older population in so many European countries. International agencies and Member States increasingly view this commitment to child and adolescent health as an investment, not as a cost. Such an investment now will bring economic and social dividends to every country, as well as to the European Region as a whole", and "Health is clearly an economic good with benefits not just to the individual, but to the wider community" [from the document for the WHO Regional Committee www.euro.who.int]. The above quoted statements taken from WHO documents highlight the importance of adolescent health and welfare issues in both scientific research and global and regional health policy.

The issues of adolescent health and well being are also an essential part of the European public health policy. They gained even more drive with the European Commission's *White Paper on Youth* of 2002. The document has become a new framework for European cooperation in the area of adolescent health. This cooperation has resulted in the development of new financial and legal instruments to support scientific research on adolescent health and well-being and health programmes. In many countries, including Poland, health of adolescents has been one of the most neglected and marginalised areas of the public health policy in recent years.

The adolescent stage of life and its health and quality of life issues have been subject of extensive study in the project entitled "At the doorstep to adulthood: adolescent health and quality of life in a variety of socio-economic backgrounds" ADOPOLNOR. The project was implemented in the years 2008–2011 at the Adam Mickiewicz University, Poznań, Poland, in cooperation with the Karol Marcinkowski University of Medical Sciences, Poznań, Poland, and Universitetet i Agder, Kristiansand, Norway. The research work was supported by the Research Funds 2008–2011 and co-financed by a grant from Iceland, Liechtenstein and Norway through the EEA Financial Mechanism and the Norwegian Financial Mechanism under the *Academic Research* priority sector.

The aim of the study was to draw a comprehensive profile of physical growth and general health, subjective well-being and health-related quality of life of adolescents, aged 10–18 in the Wielkopolska region in relation to the socio-economic status of their families and their own health-related behaviour.

Various biological and health-related as well as societal outcomes of the study serve to illustrate the interactions of somatic, physiological, functional, behavioural and societal factors that outline unique features of the adolescent period of life.

This book, scheduled primarily for scientific publication, considers adolescent health and well-being holistically, integrating biological, emotional and behav-

ioural, medical and social dimensions and takes a developmental perspective across the life course as adolescence is only one of the stages constituting the way to adulthood. The design of the book is aimed at appealing to human auxologists, epidemiologists, medical doctors, physical culture and sport researchers, health educators, practicing health professionals, and interested lay readers alike.

The book is divided into four main sections (1) Health and Environment, (2) Physical Growth and Fitness, (3) Quality of Life, (4) Health Problems and Chronic Conditions. Successive chapters of each section take up each of the major ways of contemporary research into adolescent health starting with health-related factors of physical and social environments, through the variation in normal physical growth, fitness and physiological capacity, the quality of life and ending with ill-health, needs for health care and treatment in the region.

The book begins with a carefully reasoned analysis of the multifaceted meaning of health and special health risks arising during the adolescent stage of life. The conceptual framework for the study of health marks out both positive (including normal physical growth and development) and negative (chronic conditions, disabilities) health indicators and their environmental (physical, social and cultural) determinants.

The first chapter describes a variety of concomitant factors originating from natural, built and socio-economic environments of the Wielkopolska province that determine adolescent health. Both uni- and multi-variate analyses of those factors and their mutual interactions allowed identification of those areas of the Wielkopolska region that show a high risk of morbidity.

Four chapters involved in the section devoted to physical growth and fitness provide information about variation in normal growth and development of somatic and physiological traits based on cross-sectional survey conducted in a 10 to 18-year cohort of males and females from the Wielkopolska region. Percentile values and growth charts have been drawn and are recommended as reference tools for assessing variation in somatic growth, physical fitness and distribution of arterial blood pressure across age groups of adolescents. The young people's lifestyles depended on their families' social status are presented in the chapter on social biological status differences and health disparities.

The section Quality of Life includes two chapters. The first one shows findings concerning the quality of life for males and females aged 13–18, based on the Polish version of the Youth Quality of Life questionnaire. The other provides readers with findings on perception and evaluation of own body image. It appeared that the level of satisfaction with body size was most strongly influenced by gender and BMI as well as lifestyle variables. SES conditions did not have any significant impact on the level of satisfaction with one's own body.

Four chapters of the next section provide information on health status based on secondary sources of information. Accidents and injuries were the most frequent causes of deaths among adolescents. Allergies, permanent musculoskeletal disorders and deforming dorsopathies were the most frequently recorded health problems in young people. Congenital cardiac and musculoskeletal anomalies proved to be of the highest incidence of all congenital malformations. Leukaemia as well as

malignant brain and central nervous system neoplasms accounted for the highest proportion of neoplasm cases in children and adolescents.

The most significant health care needs in the region of Wielkopolska are presented in the chapter on long-term and chronic conditions in adolescents with particular consideration to cardiovascular diseases based on medical examinations carried out under the project.

The next two chapters provide data on molecular epidemiology and genetic tests. Molecular tests were used to identify the presence of herpes viruses type I (HSV1) and type II (HSV-2), cytomegalovirus (CMV) and human papillomavirus (HPV). This pioneer research is particularly relevant to HPV, and in the future possibly also to HSV vaccination programmes. The results of genetic tests and analysis of polymorphism in the regulatory region of P1 promoter of *IGF-1* gene series may prove helpful in diagnosing growth and cardiac disorders.

Considering the multifaceted of the adolescent health topic, it is not surprise that such a book cannot cover all issues. Despite the selective nature of this book, we are hopeful to offer a new insight to anthropological, medical and sociological dimensions of human adolescence. We hope that our message will last in the lively minds of scholars, parents, teachers, guardians and adolescents.

## References

Bogin B.: *Patterns of Human Growth*. 2nd ed. Cambridge University Press, Cambridge 2005.

Bielicki T., Szklarska A.: Secular trends in stature in Poland national and social class – specific. *Ann Hum Biol* 1999; 3: 251–258.

Chassin L., Hussong A.: *Adolescent Substance Use. Handbook of Adolescent Psychology*. John Wiley & Sons, Inc. 2009. DOI: 10.1002/9780470479193.adlpsy001022.

Cole T.J.: Secular trends in growth. *P Nutr Soc* 2000; 59: 317–324.

Danker-Hopfe H.: Menarcheal age in Europe. *Am J Phys Anthropol* 1986; 29(S7): 81–112.

DiClemente R.J., Santelli J.S., Crosby R.A.: *Adolescent Health: Understanding and Preventing Risk Behaviors*. John Wiley & Sons 2009.

Garber J.: Depression in Children and Adolescents. Linking Risk Research and Prevention. *Am J Prev Med* 2006; 31(6S1).

Gluckman P.D., Hanson M.A.: Evolution, development and timing of puberty. *Trends Endocrinol Metab* 2006; 17: 7–12.

Hall G.S.: *Adolescence: Its psychology and its relations to physiology, anthropology, sociology, sex, crime, religion, and education*. New York: Appleton, 1904.

Hawkins J.D., Weis J.G.: The social development model: an integrated approach to delinquency prevention. *J Primary Prevent* 1985; 6(2): 73–97.

Heaven P.C.L.: *Adolescent Health The role of individual differences*. London: Routledge, 1996.

King K., Molina B., Chassin L.: Prospective relations between growth in drinking and life stress over adolescence. *J Abnorm Psychol* 2009; 118(3): 610–622.

Lear D.: Sexual communication in the age of AIDS: The construction of risk and trust among young adults. *Soc Sci Med* 1995; 41(9): 1311–1323.

Malina R.M., Bouchard C., Bar-Or O.: *Growth, Maturation and Physical Activity*. 2nd ed. Human Kinetics; 2004.

Metcalfe N.B., Monaghan P.: Growth versus lifespan: perspectives from evolutionary ecology. *Exp Gerontol* 2003; 38(9): 935–940.

Moore S., Rosenthal D.: The social context of adolescent sexuality: Safe sex implications. *J Adolesc* 1992; 15: 415–435.
Murray E., Burns J., See Tai S., Lai R, Nazareth I.: *Interactive health communication applications for people with chronic disease*. Cochrane Database Syst Rev 2005;1: CD004274.
Muuss R.E.: *Adolescent behavior and society: A book of readings* 4th ed. New York: McGraw–Hill, 1990.
Olszewska E., Łaska-Mierzejewska T.: Unemployment in the Polish countryside and its effect on the development and rate of maturation of rural girls. *Anthropol Rev* 2008; 71: 33–42.
Sawyer S.M., Drew S., Yeo M.S., Britto M.T.: Adolescents with a chronic condition: challenges living, challenges treating. *Lancet* 2007; 369: 1481–1489.
Swarr A, Richards M.: Longitudinal effects of adolescent girls'pubertal development, perceptions of pubertal timing and parental relationships on eating problems. *Dev Psychol* 1996; 32: 636–642.
Steinberg L.: Risk-taking in adolescence: What changes, and why? *Ann N Y Acad Sci* 2004 1021: 51–58.
Steinberg L.: Risk taking in adolescence. New perspectives from brain and behavioral science. *Curr Dir Psychol Sci* 2007; 16(2): 55–59.
Steinberg L., Lerner M.: The scientific study of adolescence. A brief history. *J Early Adolescence* 2004; 24(1): 45–54.
Tanner J.M.: *Growth and Adolescence*. 2nd ed. Blackwell Scientific Publications, Oxford 1962.
Tanner J.M.: *A History of the Study of Human Growth*. Cambridge: Cambridge University Press, 1981.
Tanner J.M.: The growth process. *Compr Physiol* first published 1999, published online: January 2011, DOI: 10.1002/cphy.cp070501. Accessed March 15, 2011.
*UNFPA. The state of the world population, 2003. Making one billion count: investing in adolescents' health rights*. UNFPA, New York 2006.
Wolański N.: *Rozwój biologiczny człowieka*. Wydanie: siódme, zmienione. Wydawnictwo Naukowe PWN 2006.
Wolański N.: *Ekologia człowieka. Podstawy ochrony środowiska i zdrowia człowieka*. T. 1, *Wrażliwość na czynniki środowiska i biologiczne zmiany przystosowawcze*. Wydawnictwo Naukowe PWN 2008.
van der Veen W.J.: *The Small Epidemiologic Transition: On Infant Survival and Childhood Handicap in Low Mortality Countries*. Amsterdam: Rozenberg, 2001.
Varraso R., Siroux V., Maccario J., Pin I., Kauffmann F.: Asthma severity is associated with body mass index and early menarche in women. *Am J Respir Crit Care Med* 2005; 171: 334–339.
*WHO 10 facts on adolescent health September 2008* Available at: http://www.who.int/features/factfiles/adolescent_health/facts/en/index.html. Accessed February 10, 2011.
*White Paper on Youth*. Available at: http:// www.europa.eu. Accessed February 12, 2011.
*World Population Prospects: The 2008 Revision*. United Nations, Department of Economic and Social Affairs, Population Division. Available at: www.esa.un.org/unpd/wpp2008/index.htm. Accessed March 15, 2011.
*World Youth Report 2007: Young People's Transition to Adulthood – Progress and Challenges*. United Nations, Department of Economic and Social Affairs: February 2008.

## Acknowledgments

This book benefited from the invaluable contribution of many people. Thanks go to the project researchers whose knowledge and research experience proved to be of great value at every stage of the study.

This project could not have been completed without the kind assistance of school directors and youths' parents. Many thanks go to them.

Sincere thanks are due to doctors, nurses and teachers for their cooperation during survey visits at schools. Special thanks are due to young girls and boys for their participation in the study and their excellent collaboration with researchers, doctors, nurses, teachers and pollsters.

Sincere thanks are due to the Rector of the Adam Mickiewicz University (AMU), prof. dr hab. Bronisław Marciniak for stimulating attitudes towards project researchers. Researchers are also very grateful to the Rector of Poznań University of Medical Sciences (PUMS), prof. dr hab. Jacek Wysocki and the Rector of Agder University (UiA) Prof. Dr. Torunn Lauvdal for their partnerships in the project.

Above all, special and warm thanks are due to Prof. Dr. Philos. Dr. h.c. mult. Ernst Håkon Jahr, the Norwegian Coordinator of the ADOPOLNOR research project and member of the Steering Committee, whose commitment in creating Polish-Norwegian collaboration was invaluable.

Thanks go to prof. dr hab. Zenon Kokot, the vice-Rector of PUMS and the late Prof. Dr. Per Kristian Egeberg for their valuable work in Steering Committee.

Researchers are deeply grateful to prof. dr hab. Jacek Guliński, vice-Rector of the AMU whose support on each stage of the project was more than helpful.

Researchers would also like to express their appreciation to the Dean of the Faculty of Biology, prof. dr hab. Bogdan Jackowiak, for his kind support while organizing scientific seminars under the project.

Researchers kindly thank Ms Katarzyna Michalska and Ms Agnieszka Drogowska from the Ministry of Science and Higher Education and Ms Karolina Centomirska from the Ministry of Regional Development for their useful assistance in project management.

Special thanks are due to Ms Beata Hildebrandt, Ms Emilia Ciecierska, Ms Anna Wieczorek and Ms Agnieszka Zboralska from the Adam Mickiewicz University for their excellent administrative assistance.

Last but not least, thanks are due to the reviewer of this book for his critical comments and stimulating input to the authors.

Supported by research funds 2008–2011 and co-financed by a grant from Iceland, Liechtenstein and Norway through the EEA Financial Mechanism and the Norwegian Financial Mechanism.


Maria Kaczmarek

# Conceptual frameworks and methodological tools for multidisciplinary approach to the adolescent health research

**Abstract:** This chapter contains information on the research methodology used in the project ADOPOLNOR. Theoretical background refers to the evolutionary perspective on human growth and development, specific features of human puberty, variation in pubertal status and pubertal timing, and the phenomenon of health paradox of adolescence. The research recognizes the socioeconomic and cultural impacts on health-outcomes in adolescence period of life. Aiming at understanding the relationship among the variables studied, a biocultural model has been used. This model enables us to determine and evaluate interactions among socioeconomic (SES) and environmental factors, and lifestyle behaviours expressing cultural identity which alter health status through the effects of poor SES and health-risk behaviour. The second part of the chapter contains a description of the methodology applied for solving research questions, including the ethical and legal framework of the research, population and sample, study design, data collection and data analysis.

**Key words:** evolutionary perspective, biocultural model, puberty, socioeconomic status, lifestyle behaviour, health outcomes

## Unique pattern of human life cycle

The human life cycle stands in sharp contrast to other species of highly social mammals and other primates by a distinct set of features. *Homo sapiens* is characterized by a long lifespan, large brain growing rapidly during gestation period and after birth, gives birth to large babies, has a relatively high fertility with inter-birth intervals of about 2.5–3.5 years, shows strong cultural influence on reproductive decisions throughout reproductive period of life. The human baby is unusually altricial (helpless), breast fed for a short period of time, grows and develops slowly with a delayed reproduction and long periods of juvenile dependence on extensive parental care since the transition from childhood to adulthood lasts extremely long. More than any other primates, humans show an extended period of intensive parental care including large transfers of information. The human females are

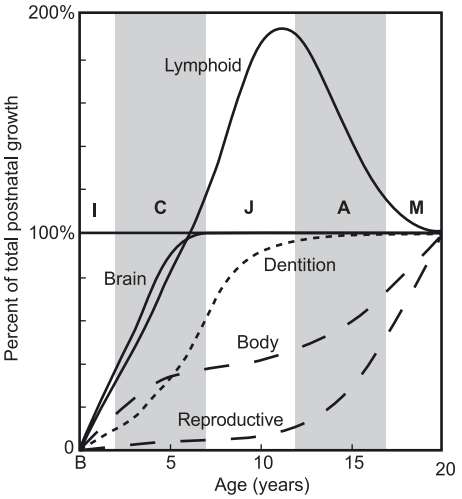



Fig. 1. Growth curves for different body tissues against the five stages of human postnatal life

somewhat unique among primates in that they have concealed (cryptic) ovulation, experience a sudden cessation of fertility and the onset of menopause followed by a long post-reproductive period of life [Bogin 2005; Flinn et al. 2007].

Flinn and colleagues [2007:16] specify some other unique features such as "…habitual bipedal locomotion, use of the upper limbs for handling tools, including throwing projectile weapons, culture including language, and lethal competition among kin-based coalitions."

Based on the rate of growth, the onset of sexual maturation and changes in trophic and reproductive behaviours, the human life cycle can be described as a sequence of five postnatal stages, including infancy, childhood, juvenility also termed juvenescence, adolescence, and adulthood [Bogin 2005:55] (Fig. 1).

In contrast, the majority of mammals commonly progress from infancy to adulthood directly without any intervening stages and puberty occurs while growth rates are in decline [Bertalanffy 1960; Tanner 1962; Coehlo 1985] (Fig. 2).

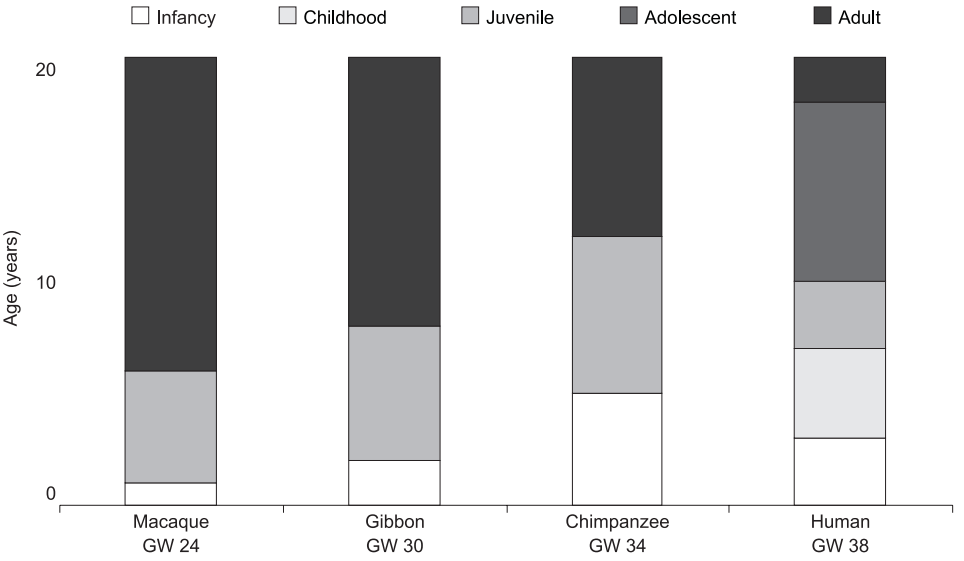



Fig. 2. The main life stages in macaque, gibbon, chimpanzee, and human. GW – an average pregnancy length in gestation week

Human adolescence is a part of human life cycle, the transition period between childhood and adulthood, during which physical changes interfere with substantial brain maturation resulting in acquisition and refinement of the physical, cognitive, emotional and social skills that will allow young people to live independently of their parents, successfully reproduce and become parents. This period is marked by typical physical and behavioural changes. These changes result from interplay of biological and environmental cues. The biological factors arise from the genetic, hormonal and neural domains. The potential role of the so-called clock genes and the cumulative effects of hormonal and neural maturational cascades in the process of generating irreversible developmental states cannot be overestimated [James et al. 2007]. The environmental factors typically arise from an individual's living conditions, in particular nutritional conditions, her or his exposure to estrogenic or antiandrogenic agents and many others.

Modern concept of human adolescence encompasses a biological onset and highly variable social-role passages that mark its completion. The biological processes initiated at puberty interact with social context variables to affect an individual's cognitive and psychosocial development. Association of major physical growth with substantial brain maturation during adolescence is a unique feature for humans that distinguish them in the animal world [Spear 2004].

## The paradigm of puberty

Human puberty is typically initiated at the beginning of the second decade of life with subtle changes in neuroendocrine processes, physical (morphological and physiological), psychological and behavioural characteristics. These changes are considered to result from dynamic interaction between genetic factors and environmental cues; they are marked by the development of secondary sexual characteristics, accelerated growth, and behavioural changes ultimately leading to the attainment of reproductive capacity and psychosocial maturation [Tanner 1989: 380]. Puberty marks the transition from the sexually infantile and non-reproductive state into complete sexual maturation and reproductive state of life which "…requires a phenotypic switch, to coordinate the appropriate suite of traits and behaviors in an adaptive fashion", first at the passage from childhood to juvenility and then when juvenility turns to adolescence [Del Giudice et al. 2009: 2]. Both onset and trajectories of physical (morphological and physiological) changes that characterize puberty are well documented. Puberty as a social construction is a more complicated concept and entails definitional ambiguity regarding social-role passages into new reference groups, perceived body image and self-esteem, and expectations for independent and mature behaviour [Alsaker 1995].

The phenotypic changes, which occur in adolescence, are considered to result from two independent, though related by a factor of energetic status, neurohormonally driven processes: adrenarche and gonadarche [Tanner 1962; Wolański 2006].

Adrenarche (also called adrenal activation) refers to the maturation of the hypothalamic-pituitary-adrenal (HPA) system and the zona reticularis of the adrenal gland, resulting in increased secretion of adrenal androgens namely dehydroepiandrosterone (DHEA), dehydroepiandrosterone sulphate (DHEA-S), and androstenedione [Tanner 1962; 1989].

Adrenarche is a unique phenomenon confined to humans and some other higher primates [Arlt et al. 2002]. There is some evidence that adrenarche occurs before puberty in chimpanzees, and during the neonatal period in rhesus macaques and baboons [Conley et al. 2004]. The exact mechanism that triggers the adrenarche remains unknown, however it has been suggested that alterations in the utero and neonatal physiology, including adrenocorticotropic hormone (ACTH) and/or 3-hydroxysteroid dehydrogenase may modulate this physiologic phenomenon. The postnatal onset of adrenarche is marked by increased 17$\alpha$-hydroxylase and 17,20 lyase activity of the P450c17 enzyme and increased cytochrome b activity resulting in increased DHEA, DHEA-S, and androstenedione production, and maximum increase in body mass index (BMI) [Remer 2000; Ong et al. 2004]. These initial hormonal increases rise over time, resulting in a cumulative dosage of androgens.

In humans, adrenarche typically begins between 7 and 8 years of age in both genders, but the timing of clinical signs can vary and in some cases can occur as early as 6 years [Tanner 1989; Ibanez et al. 2000; Auchus et al. 2004]. In both genders, adrenarche results in the appearance of pubic and axillary hair (pubarche), body odor and occasionally the appearance of acne and the adiposity rebound at the transition between the childhood and juvenile stages of the life cycle [Tanner 1989]. There is also evidence suggesting that the timing of adrenarche might have implications for both adolescent health problems and illnesses later in adulthood [Golub et al. 2008].

Gonadarche, often overlapping adrenarche or occurring 1 to 2 years later, is essentially the most important mechanism that brings about a complex series of developmental and neuroendocrine events that lead to full activation of the gonadotropin-releasing-hormone (GnRH) pulse generator, enhanced gonadotropin secretion and complete gonadal maturation and function. Gonadarche involves maturation of the hypothalamic-pituitary-gonadal (HPG) system. This mechanism controls the development of the secondary sexual characteristics, maturation of the genitalia and the ability to produce mature gametes (sperms or oocytes) capable of fertilization [Grumbach 2004]. At adolescence, hormones from the gonads combine with growth hormone (GH) and produce the adolescent spurt. Individual differences in the tempo of growth have substantial implications on major cognitive, emotional and social skills of adolescents as evidenced from developmental studies [Sisk and Foster 2004].

Recent progress in genetic, genomic and physiological studies in primates and other mammals, in combination with bioinformatics strategies and a systems biology approach, has allowed one to understand major aspects of the human reproductive biology and puberty in particular [Ojeda et al. 2010; Roa et al. 2011]. The neural control of puberty embraces the timing of puberty and the mechanisms in-

volved in the control of the phenotypic switch between prepubertal, sexually neutral state and complete sexual maturation attained during puberty.

Schematic representation of the regulation of human puberty onset and progression via the HPG and the HPA axes is shown in Figure 3.

Puberty viewed from an endocrine perspective refers to the activation of the hypothalamic-pituitary axis and its consequences on structure and function of brain, physical growth and sexual maturation [Tanner 1962; Sisk and Foster 2004]. It is also considered to be a developmental milestone in the continuous process of growth and maturation that involves the excitation of the hypothalamic GnRH pulse generator and gonadotropin secretion after the period of quiescence during childhood. The gradual rather than abrupt pattern of changes means that puberty is not a fixed process; it can be arrested or even reversed [Grumbach 2002:10].

Human studies, using peripheral levels of LH and FSH as indices of GnRH activity and animal models that permit direct assessments, have revealed its functional onset at early stages of fetal life and its activity for at least 0.3 gestation. GnRH has been detected in human embryonic brain extracts and in the fetal hypothalamus as early as 4.5 weeks of gestation. The GnRH neurons have been demonstrated in the fetal hypothalamus by 9 weeks gestation, although functional con-

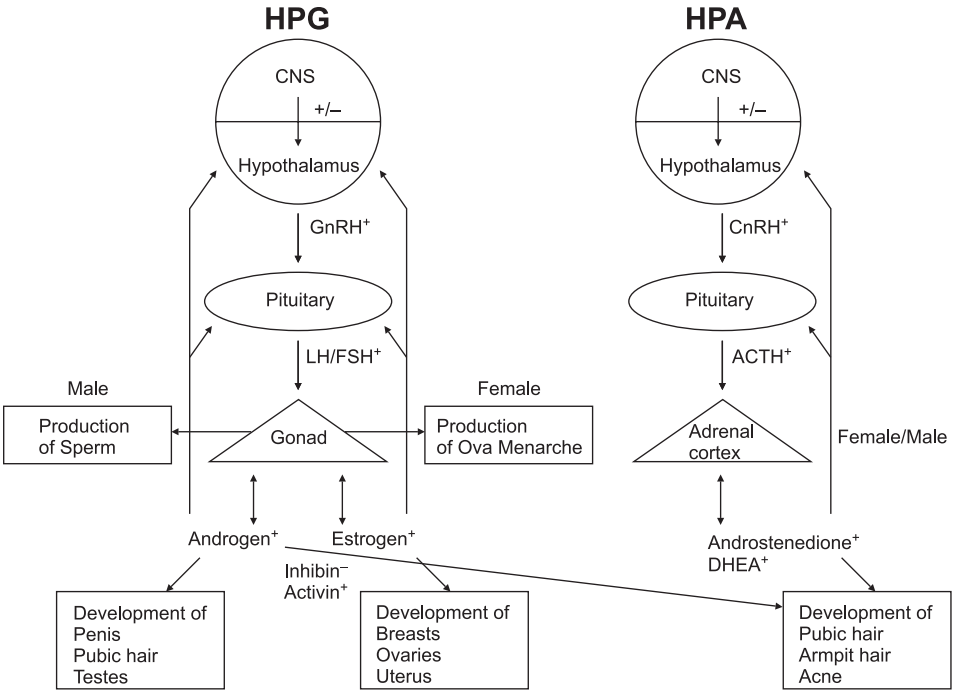



Fig. 3. Schematic representation of the HPG and the HPA axes regulating the onset and progression of human puberty (adrenarche and gonadarche). Modified from G.M. Buck Louis et al. 2007; 121: S193

nections between these neurons and the portal system are not established until 16 weeks. The serum LH and FSH levels are first detectable in the pituitary at 10 weeks gestation and are measurable in the fetal circulation by 12–14 weeks, then rise gradually to a peak at about 20 weeks of gestation and fall to low levels by the time of birth associated with the negative feedback effects of steroids on the hypothalamo-pituitary axis.

During the neonatal period, there is clear evidence of GnRH secretion as evidenced by elevated level of circulating FSH and LH with peak around 2–3 months of postnatal age and gradual decrease in its activity by about 6 months of age in boys and 12–24 months of age in girls. During the childhood years, the HPG axis remains quiescent until the onset of puberty; this is the so-called juvenile pause evidenced by low prepubertal levels of gonadotropins. The gradual disinhibition and reactivation of the GnRH oscillator (mainly at night) occur during late childhood, and is reflected in the progressively increased and changing pattern of circulating LH pulses, with the approach of and during puberty [Grumbach 2002:2; Styne and Grumbach 2007]. During the juvenile phase, the intrinsic central nervous system (CNS) inhibitory neurotransmitter γ-aminobutyric acid (GABA), GABAergic and opiatergic neurons provide the most important inhibitory inputs to the GnRH pulse generator and restrained activation of the hypothalamic-pituitary-gonadal system [Terasawa et al. 2010].

The initiation of human puberty is heralded by an increased pulsatile nocturnal release of gonadotropin releasing hormone (GnRH) produced by a network of peptidergic neurons in the medial basal hypothalamus, which acts as an endogenous pulse generator (oscillator). Reactivation of the GnRH puls generator is associated with a fall in GABAergic neurotransmission and concurrent increase in the input of excitatory amino acid (EAA) neurotransmitters and possibly glial cells via growth factor-dependent cell-cell signaling. The pituitary gonadotropes, in response to increased pulse amplitude and possibly increased pulse frequency of the GnRH signal, release luteinizing hormone (LH) and follicle-stimulating hormone (FSH) in a pulsatile manner [Wu et al. 1996; Mitamura et al. 1999; Terasawa and Fernandez 2001]. The gonadotropins in turn, bind to ligand-specific receptors in the gonads, the ovaries in females and testes in males, to stimulate gonadal growth and maturation: gametogenesis – production of haploid sex cells (ovum and sperm) and steroidogenesis – production of the gonadal steroid hormones specific to each sex, a male testosterone and a female estradiol [Delemarre-van de Waal 2002; Tena-Sempere and Huhtaniemi 2003; Tena-Sempere 2006]. These three levels of the hormonal network comprise the so-called gonadotropic axis also termed hypothalamic-pituitary-gonadal (HPG) axis, which is primarily composed of three major hierarchical elements: the hypothalamic GnRH, the pituitary gonadotropins (FSH and LH), sex steroids and other hormones produced by the gonads that function either independently or interdependently.

Diagrammatic representation of the physiological paradigm of puberty is shown in Figure 4.

While the mechanism(s) that triggers the onset of puberty is still unclear, recent studies have suggested that pubertal reactivation of GnRH secretion is

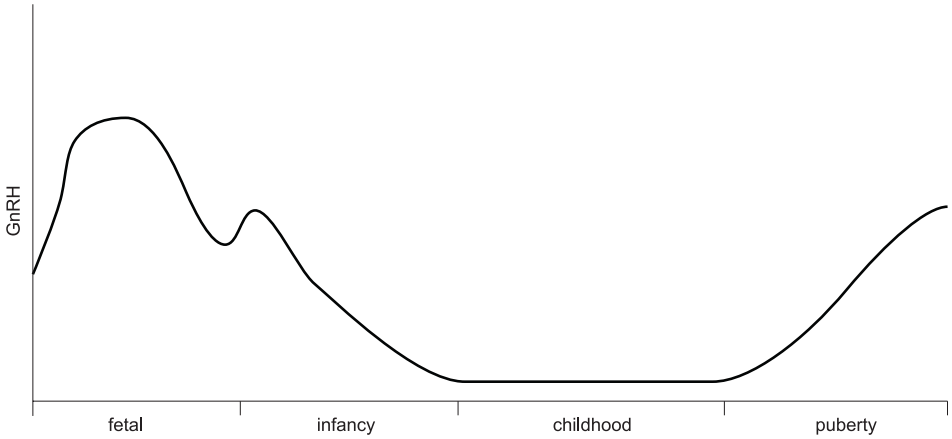



Fig. 4. GnRH secretion from fetal development into adulthood; gonadal quiescence typical for prepubertal development is marked by prepubertal restraint

brought about by a number of permissive signals i.e. coordinated changes in transsynaptic and glial-neuronal communication. Two of these events are transsynaptic (a decrease in GABAergic inhibition and an increase in glutamatergic stimulation to GnRH neurons) and the third involves activation of a reciprocal glial-neuronal communication pathway [Styne and Grumbach 2007; Ojeda et al. 2010]. The release of GnRH may also be facilitated through the adipose derived factor leptin, peripheral hormones (the gonadal steroids *inter alia*), and metabolic cues [Tena-Sempere and Barreiro 2002]. There is evidence of multiple signals, the so-called "permissive" signals that permit puberty to occur or progress, but the precise causes remain unknown.

New light was shed on this issue when in 2001, kisspeptins (KP), the peptide products of the *KiSS-1* genes translation, were identified as natural ligands of the G protein-coupled receptor GPR54 [Kotani et al. 2001; Muir et al 2001; Ohtaki et al. 2001].

Genetic, physiological, pharmacological and clinical data, mostly obtained in studies of nonhuman and rodent models, have demonstrated that levels of KiSS-1 and GPR54 mRNA increases dramatically at puberty, suggesting that kisspeptin signaling could mediate the GnRH secretion, which is the key event that triggers the onset of puberty [Colledge 2004].

It has been proven that the hypothalamic KiSS-1/GPR54 system is an essential gatekeeper of GnRH neurons which allows for the integration of central and peripheral inputs, thereby playing a fundamental role in the neuroendocrine regulation of pubertal timing, its proper course and initiation of the pulsatile secretion of LH and FSH hormones. Coordination of this highly complex and intricate hypothalamic mechanism that controls puberty requires sets of genes hierarchically arranged within functionally connected network that includes GPR54. Epigenetic mechanisms may provide transcriptional plasticity to this genetic network.

As puberty approaches, the hormonal regulation of physical growth, psychological and emotional development and sexual maturation becomes increasingly com-

plex. It includes a striking activation of the growth hormone-insulin like growth factor (GH/IGF-1) and thyroid axes, which in interaction with the sex steroid hormones lead to the pubertal growth spurt, skeletal maturation and achievements of adult height, changes in body composition, maturation of many organ and enzyme systems, and the development of secondary sex characteristics [Veldhuis 2003]. Clinical observations have shown that normal pubertal growth is mediated through both GH and gonadal steroid hormones under condition of sufficient levels of estrogen. Many of the growth-promoting effects are mediated through estrogens including skeletal maturation and ultimate fusion of epiphyseal plates.

Puberty is also associated with peak level of risk-taking behaviours that characterize adolescent motivated behaviour and alteration of the social landscape. Risk-taking behaviour, often understood as challenges, refers to undertaking a task that has the potential to be harmful or dangerous, yet at the same time provides the opportunity for some kind of outcome that can be perceived as positive. From an evolutionary perspective, human risk-taking behaviours should be viewed in relation to evolutionarily recurrent survival and reproductive problems [Kruger et al. 2007]. Accordingly, these novel or slightly dangerous behaviours are essential to facilitate the separation of the adolescents from their families and launching an independent life, avoidance of genetic inbreeding and spread of genetic diversity. As such, adolescent behavioural changes allow organisms to meet recurring environmental challenges to their survival, thereby increase their reproductive ability. Risky behaviours seem to be a normal part of adolescence, an important tool for discovering, developing, and consolidating adolescents' identity. Unfortunately, it comes with a cost due to experiments and riskiness that could impact on adolescents' own health and wellbeing and have consequences for others. These risky behaviours typically manifest as antisocial behaviour (physical aggression, relational aggression, behaviour problems, delinquent and violent behaviour, substance use and abuse, risky sexual activity, and others); reflect unmatched rates of morbidity (conduct disorder symptoms, mental health disorders and others), and rate of mortality (mainly from accidents and suicides).

Different hypotheses have been postulated for understanding the adolescent risk-taking behaviour. These hypotheses, build on animal models and structural and functional Magnetic Resonance Imaging (MRI) as well as other techniques of human brain imaging, emphasize the complexity of the morphological/anatomical and functional organization of neural and endocrine systems underlying this behaviour. The hormonal model postulates long-lasting consequences of gonadal steroid hormones, which levels become elevated during puberty, on brain structure and function, sexual drive, developmental trajectory and behavioral maturation [Sisk and Zehr 2005].

The steroid hormone testosterone has been implicated in physical aggression in animals. In humans, and particularly in adolescents, findings have been less consistent, suggesting that this may partly be due to moderating effects of other hormones, e.g. cortisol [Popma et al. 2007]. It was found a positive relation between testosterone levels and aggressive behaviours in humans [Archer 2006]; sexual maturation and substance use culminating in a substance use disorder (SUD) in

adolescent males [Reynolds et al. 2007]; coitus in females [Halpern et al. 1997], and sexual activity in males [Halpern et al. 1998]. A negative relation was found between testosterone and behaviour problems in healthy young boys [Susman et al. 1987].

As Sisk and Foster [2004:1045] claim, "Neuroscientists have made enormous strides in calling attention to the role of the brain in reproductive maturation, in identifying the proximal signals and neural mechanisms that drive the activation of GnRH neurons at the onset of puberty, in characterizing the neural circuits as a time of profound remodeling of the brain".

The adolescent motivated behaviour, viewed from neural perspective, has been suggested to be governed by well organized articulation among three systems: approach, avoidance and regulatory that are mapped to distinct, but overlapping, neural circuits, whose representatives are the striatum, the cerebellum amygdala and the medial prefrontal cortex [Ernst and Fudge 2009: 367]. The Triadic Model, postulated by Ernst and Fudge, corresponds well to behavioural, clinical, and neurobiological evidences reviewed by Casey and Jones [2010]. The reviewed studies have suggested that the mechanism underlying adolescent motivational and cognitive behaviours is characterized by imbalanced interactions between two regions, the striatum, subcortical brain regions (bottom-up systems) and the prefrontal cortex (top-down systems) [Casey and Jones 2010: 1189]. The striatum regions typically mature earlier than the prefrontal cortex. During adolescence, motivational cues of potential reward are particularly salient and can lead to hazardous choices that diminish effective goal-oriented behaviour. The reasons why adolescents can have difficulty controlling their emotions and impulse control are complex; they are the result of brain immaturity. Somerville and Casey [2010] have drawn cartoon model illustrating developmental trajectories for signaling of these regions. In the model, the motivational behaviour and the underlying subcortical brain regions, important in the bottom-up regulation of behaviour, show curvilinear developmental pattern and peak inflection from 13 to 17 years. In contrast, prefrontal regions, important in the top-down regulation of behaviour, show a linear pattern of development [Somerville and Casey 2010: 237; Casey and Jones 2010: 1197]. These developmental patterns vary across individuals. That is, although adolescents as a group are considered risk-takers, some adolescents will be more prone to recklessness and risk-taking behaviours than others. This finding may help the health policy maker to create more constructive strategic programmes for reducing the adolescent harmful risk-taking opportunities.

## Variation in the status and timing of puberty

The basic patterns of physical development in adolescents are universal but their status (the adolescent's current level of physical maturation) and timing (pubertal status relative to the same-age peers; whether one matures on time, early, or late) are extremely variable. The critical meaning of the timing is that the individual

must perceive, through metabolic cues, whether its growth is sufficient; through social cues what its relationship is to other individuals, and through environmental cues whether conditions are optimal to begin the reproductive process.

The onset of puberty is heralded by physical changes that result from the combination of adrenarche and gonadarche. It typically corresponds to a skeletal age of approximately 11 years in girls and 13 years in boys. On average, girls enter and complete each stage of puberty 2 years earlier than boys, but there is significant individual variation in the timing and tempo of puberty.

The normal range of onset of puberty is ages 8 to 14 in girls and 9 to 15 in boys. The 4–5 year variation in age at onset of puberty is a physiological peculiarity of the human species observed among healthy individuals, despite relatively similar life conditions [Tanner 1962]. This variation reflects a strong genetic component, as indicated by the studies on heritability of menarcheal age [Kaprio et al 1995]. Other factors such as nutritional conditions, psychological status, socioeconomic conditions and secular trends have shown some additional effects [Bielicki and Welon 1982; Eveleth and Tanner 1990; Hulanicka et al. 1994; Wolański 2008]. Pathological pubertal precocity, most commonly reported by females, is associated with premature activation of the HPG axis and pubertal delay with chronic illness, stress, and malnutrition.

Puberty is a time of significant height and weight gain. Given growth data for young Poles, one may see that the prepubertal growth velocity height declines progressively with age reaching a nadir (preadolescent dip; in velocity curve denoted as the take-off (TO) at average age 8.9 in girls and 10.8 in boys with corresponding attained height 133.8 cm and 143.9 cm in girls and boys, respectively and growth velocity 5.2 cm/year in girls and 4.8 cm/year in boys [Kaczmarek 2001]. During mid-puberty, the growth velocity suddenly accelerates (the pubertal growth spurt) and attains its peak. Girls attain a peak height velocity (PHV) of 7.4 cm/year on average at age 11.8 when breast are at stage 3. Boys average a peak height velocity of 9.3 cm/year two years later than girls, at age 13.9 [Kaczmarek 2001].

Peak weight velocity (PWV) averages 5.6 kg/year and 6.6 kg/year in girls and boys, at ages 12.5 in girls and 13.9 in boys [Kaczmarek 2001]. In girls, the PWV lags behind the PHV by approximately 0.7 years. But in boys, these two events occur coincidentally. The rate of height and weight gains decelerates during the later stages of puberty until the attainment of adult size.

The marked changes in body composition, including alterations in the relative proportions of water, muscle, fat, and bone, are characteristic of pubertal development and the result of female to male differences. Boys typically accrue fat-free mass (FFM) at a much greater rate and for a longer time than girls. The young adult amount of FFM is attained at age 15 to 16 years for girls, but 19 to 20 years for boys [Malina and Bouchard 1991]. Pubertal girls increase the percentage of body fat and accrue fat mass at a rate of 1.14 kg/year. Pubertal boys decrease the percentage of body fat by 1.15 kg/year, but the fat mass remains relatively constant. The changes in the distribution of body fat result in the typical android (males) and gynoid (females) patterns of fat distribution of the older adolescent and young adult [Roemmich et al 2000].

Sexual changes occur under the influence of gonadal steroid hormones, predominantly testosterone in males and estradiol in females, and the adrenal androgens, primarily dehydroepiandrosterone sulfate (DHEAS). This process follows a regular sequence of events within each gender, but individual variation does occur normally.

In girls, the first clinical sign is the appearance of breast buds at stage 2 (thelarche – breast development) and the appearance of dark straight pubic hair over the mons pubis (pubarche). The thelarche typically occurs at the mean age of 11.0 years. Breast development over the next 4 years will proceed from breast stage 2 to adult breast stage 5. Development of pubic hair starts about one year after breast budding and may take place over a 1.5 to 3.5 year period [data for Tanner stages also called Sexual Maturation Rate (SMR) from the UK: Marschal and Tanner 1969, 1970; Tanner et al. 1975]. During Tanner stage 3, girls experience the peak of their height growth (PHV), which should take place before the onset of menarche in most girls. Menarche is reached after a series of complex developmental and neuroendocrine events leading to full activation of the HPG axis including maturation of the KiSS-1/GPR54 system. It occurs on average one year after the PHV and just prior to stage 4 of breast development. Although there is a wide range of variation among individuals in age at menarche, most western girls achieve their menarche between 12 and 13 years of age [Eveleth and Tanner 1990]. Mean age at menarche of Polish girls is 12.8 [Kaczmarek 2001; 2008 unpublished data]. Estimation of variance components has shown that the timing of menarche is a complex biological trait determined by an array of genetic and environmental factors [Anderson et al. 2007]. A review of family and twin studies has shown the timing of menarche to be a highly heritable trait with reporting heritabilities ranging from 0.44 to 0.95 [Towne et al. 2005].

Recent genome-wide association (GWA) studies have identified several novel genetic loci and candidate genes associated with age at menarche. The proposed 9 groups of candidate genes in biological pathways or for related-phenotypes include: steroid-hormone metabolism and biosynthesis pathway, insulin-like growth factor (IGF) pathway, transforming growth factor-beta (TGF-β) superfamily and signaling pathway, thrombophilia and vascular homeostasis pathway, obesity and obesity-related phenotypes, and precocious or delayed puberty [Chunyan et al. 2010: 521]. Several candidate genes associated with age at menarche that have been identified so far, include the estrogen receptor-α (*ESR1*) gene [Stavrou et al. 2002] and the estrogen receptor-β (*ESR2*) gene that are supposed to interact with each other [Stavrou et al. 2006], and the estrogenbiosynthetic gene aromatase *CYP19A1* [Guo et al. 2006].

In boys, the first sign of puberty is an enlargement of testicular volume to greater than 4 mL, usually at the mean age of 11.1 years [data for the UK from: Marschal and Tanner 1969, 1970; Tanner et al. 1975]. Enlargement of the testes indicates the transition from genital stage 1 to stage 2, beginning at an average age of 11.5 years. The complete change from preadolescent to adult takes between 2 and 5 years. Penile growth occurs about one year later. This is usually preceded by the appearance of pubic hair at the base of the phallus progressing through pubic hair stages 2 to 5. Pubic

hair stage 3 is followed by the appearance of axillary and facial hair growth. Testicular growth is completed anytime between 13.5 and 17 years of age. Growth of the penis reaches a sexual maturation rate (SMR) [Tanner] stage 5 between 12.5 and 16.5 years of age. Nocturnal emissions (wet dreams) may first appear during SMR stage 3.

It is estimated that 80% of girls begin puberty with thelarche (breast budding) and the other 20% with pubarche. The most common sequence of pubertal changes in girls is: Breast stage 2; Pubic hair stage 2; Peak Height Velocity; Breast stage 3; Pubic hair stage 3; Pubic hair stage 4; Breast stage 4; Menarche; Pubic hair stage 5; Breast stage 5.

Ninety-eight percent of boys begin puberty with enlargement of the testes. The most common sequence of pubertal changes for boys is: Testicular volume 4 mL/Genital stage 2; Genital stage 3; Pubic hair stage 2; Genital stage 4; Pubic hair stage 3; Peak Height Velocity; Pubic hair stage 4; Genital stage 5; Pubic hair stage 5.

Recent studies have shown a marked trend towards younger age in puberty onset observed in industrialized European countries, and in the US, since the mid-1900s until the middle of the 20th century, likely the result of improved living conditions, nutrition, hygiene and overall public health. The timing of menarche has declined from mean ages at menarche of 15 to 17 years reported in the mid 19th century [Tanner 1981] to recent estimates of the median age at menarche that remain relatively stable, between 12 and 13 years [Eveleth and Tanner 1990]. The rate of advance in age at menarche, as evidenced from recent European data, varies between and within populations with faster trends occurring in several eastern European countries, as for example in Poland [Łaska-Mierzejewska and Olszewska 2004] and trends that have slowed or even stopped in the majority of North-West European countries (as for example in Norway or Belgium). There is also marked within-population variability related to socio-economic status, urbanization and other environmental factors.

Secular changes in pubertal timing, as indicated by a falling average age in pubertal onset are less obvious for boys than for girls. However, several studies on specific populations showed that there appeared to be a downward trend in the onset of puberty in boys, usually related to BMI [Sørensen et al. 2010].

Ever-younger girls and boys entering puberty have been more and more delayed in taking on mature social roles and responsibilities in marriage, parenthood, and employment. This modern mismatch of biological and psychosocial transitions by more than a decade is exceptional in human history [Gluckman and Hanson 2006].

## The health paradox of adolescence

Adolescence, viewed from the health care perspective, is considered to be the healthiest period of the lifespan prior to adult declines and beyond the weakness of infancy and childhood. However, a period of life characterized by peak physical status; improvements in strength, speed, reaction time, reasoning abilities, and im-

mune function, is paradoxically accompanied by disproportionately increased rates of morbidity and mortality. This phenomenon contributes to the so-called health paradox of adolescence.

Recent pediatric data have shown that puberty is a phase of high risk for many health problems. Their primary sources are related to difficulties with control of behaviour and emotion. Increased impulsivity and risk-taking behaviours can lead to grave physical and socioeconomic detriment.

The major health concerns of adolescents include: chronic physical illness, mental health problems, dental problems, and health problems caused by health-risk behaviours such as intentional and unintentional injuries, use of tobacco, alcohol and illicit drugs, risky sexual behaviours, specific dietary habits, insufficient physical activity.

The government report on adolescents, entitled Młodzi 2011, draws their current portrait after twenty years of transformation in Poland. Generally, the vast majority of young Poles is in good health, satisfied with her/his life and has a lot of life energy. Taking a closer look on health statistics, the picture becomes more sensible and better reflects the health paradox of adolescence [Szafraniec 2011].

These statistics show that every seventh Polish teenager suffers from chronic illnesses, and over 17% of young people, subjected yearly to accidents and injuries, have required medical intervention.

Increasing incidence of cancer, cardiovascular diseases and diabetes share many risk factors related to poor dietary habits, sedentary lifestyle, overweight and obesity of young people.

Depression and neurosis affect every fourth teenagers. Nearly 8% of adolescents suffer from mental disorders as a consequence of addiction (mainly drugs). The reported elevated psychological stress levels and frequent occurrence of severe fatigue and exhaustion, depression and headaches have significantly increased among teens in recent years. A few years ago, 30–40% of young people complained about these symptoms. Since that time, the prevalence of young people suffering from these conditions has doubled.

The most common cause of deaths of individuals aged 1–19 years are injuries and poisoning, responsible for over half of deaths among children and adolescents. Traffic accidents are, by far, the leading cause of death among adolescents (almost 50% of deaths) as well as suicides (23%). These figures are similar to other European statistics (WHO 2008).

The top three causes of death among adolescents are accidents, cancer and diseases of the nervous system. Malignancies are responsible for almost 13% of deaths; diseases of the nervous system for 7% of deaths among children and adolescents aged between 1 and 19 years.

There has been an upward trend in suicide among adolescents, in recent years. In 2002, there were 55 cases of suicide among adolescences and in 2009, the number dramatically increased to 356. It is supposed that the main cause of young people's suicide is associated with failures encountered on the way to adulthood.

A growing problem is unsafe sexual behaviour resulting in HIV infection, other STDs, and unintended pregnancy. In Poland, among the HIV-infected people,

42.9% account for young adults aged 15 to 29 years. Of them, 4% account for teenagers below 19 years of age. The highest risk group among young Poles is drug addicts – they constitute more than 58% of the newly diagnosed HIV-infected people.

Another serious problem is deviant eating patterns contributing to eating disorders such serious as anorexia nervosa, bulimia nervosa, or binge eating disorder. In just a few years, the percentage of young patients treated in clinics because of this type of disorder has increased relative to the total number of patients from 16% to 45%. Individuals, who are underweight, consist of 11.5% of young Poles aged 15–24 years, with girls being underweight three times more often than boys. The overweight accounts for one in ten young people with boys outnumbering girls two to one.

The WHO data indicate that in all European countries, the initiation of alcohol use takes place between 13 and 14 years of age, almost the same age in both genders. However, young Poles are less likely to drink alcohol regularly as compared to their peers from other European countries.

Prevalence of smoking cigarettes among Polish youth is one of the highest in Europe, although a steady decline of this habit has been observed since 2000. The daily smokers among teenagers account for 29% of boys and 20% of girls. The average age of smoking initiation is 13 years for boys and 15 years for girls.

Despite an increase in rates of drug use in recent years, Poland still maintains its position below the European average. However, in consumption of psychoactive substances, Polish teenagers are on the top of European ranking.

The adolescence period is believed to be critical for long-lasting health consequences. Since the health problems of young people have their source mainly in risky behaviour, the improvement of health may be achieved through an appropriate mode of life. That is why promoting healthy practices during adolescence and efforts that better protect this age group from risks will ensure healthier, longer, more satisfying and more productive lives of people from the entire population.

## Integrated biological and sociocultural approach to the study of adolescent health and well-being

Identification and targeting of adolescents' groups at high risk for health outcomes is of great importance, given that recent studies have recognized synergistic role of sociocultural environment and human behaviour in health outocmes. Strong associations between social and behavioural problems, which either start or peak during adolescence period, and an individual's health and well-being are well established. There is evidence from population-based studies that a low poorer socioeconomic status (SES) usually creates hardship and affliction and as such is a risk factor for delayed physical growth and maturation, obesity, and chronic conditions [Bielicki and Welon 1982]. Not only standard of living but also harmful adolescent behaviour such as homicide, suicide, motor vehicle crashed caused by

drinking or driving too fast, substance use and abuse, smoking, sexually transmitted infections including HIV, teen and unplanned pregnancies, and homelessness are important elements of the causal pathways to health status.

Environmental and sociocultural determinants of health and well-being are schematically shown in Figure 5.

This diagram represents a modified version of a conceptual model proposed by Northridge et al. [2003: 559] for understanding social determinants of health and environmental health promotion.

The model posits a set of simultaneous and dynamic relationships among factors structured on three levels of the environment: macro (fundamental), meso (community) and micro (interpersonal) levels that underlie and influence health and well-being via multiple pathways. Fundamental factors, operating on macro level may influence health status and well-being through differential access to resources (including natural environment), power, and information. These factors in turn influence the intermediate level which is depicted by factors related to resources and social context of the community, including build environment (land use and buildings, transportation system, services and others) and community capacity.

Moving to the proximate level, three domains can be distinguished, including various stressors (such as violent crime, financial insecurity, and environmental

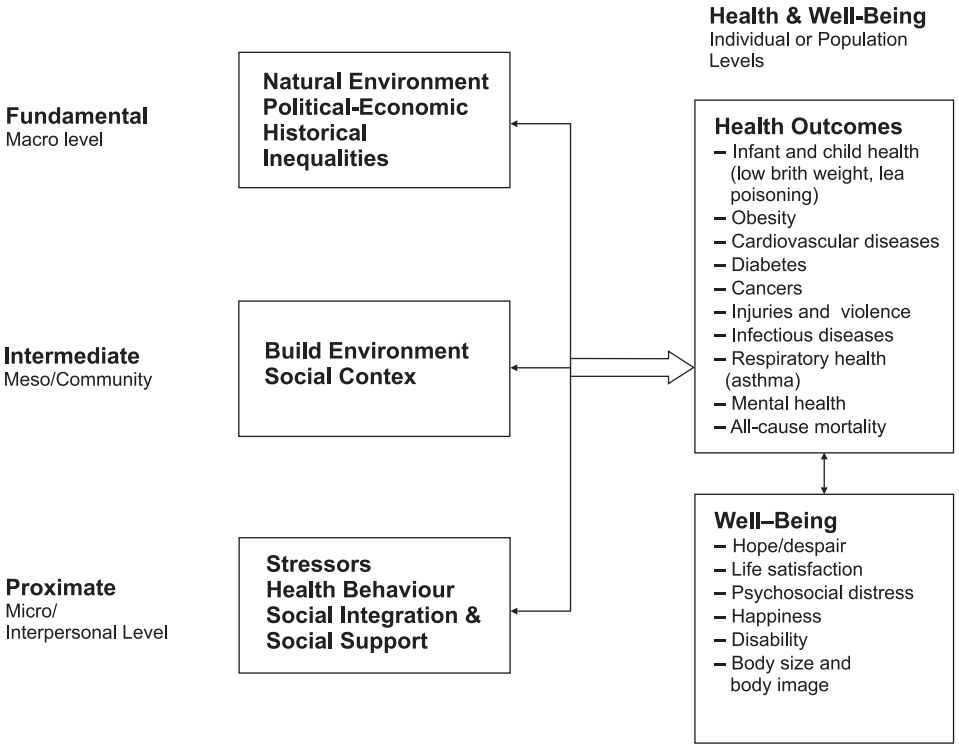



Fig. 5. Determinants of human health and well-being

toxins), and social integration and social support (including the shape of social networks and the resources available within networks). The third domain includes habits relevant to health such as dietary practices, physical activities, substance use and health screening.

Indicators of health status, depicted in this diagram, include anthropometric and physiological characteristics of physical growth and fitness, obesity, injury and violence, and chronic conditions (respiratory, cardiovascular and others). Well-being effects include life satisfaction, body image, hope/despair, disability and others.

Towards better understanding the complex relationships among adolescent health, behaviour (including lifestyle behaviour) and sociocultural environment, a theoretical framework provided by biocultural biology has been employed. Biocultural approach to human health by incorporating an ecological or multilevel perspective, integrates biological, social and cultural perspectives on human variability and adaptability with special focus on the role of sociocultural environment [McElroy 1990; Armelagos et al. 1992; Bogin and Varela-Silva 2003; Dufour 2006]. The integrated biocultural model (Fig. 6) provides a framework for understanding the dynamic interactions among human biological variation, physical, biotic and sociocultural environments, and explaining how cultural system buffers the population from insults that originate in the environment, and how cultural system may be the source of insults to the population [McElroy 1990].

Health outcomes and health risks across populations, shaped by the expression of genetic inheritance and the relative fitness of individuals, and confronting certain kinds of triggering environmental stressors, provide a base for an integrated view of human-ecosystem interactions over evolutionary time [Stinson et al. 2000]. Health and disease are considered as a continuum operating within a spectrum. The health/disease status of an individual, group or community can be ap-

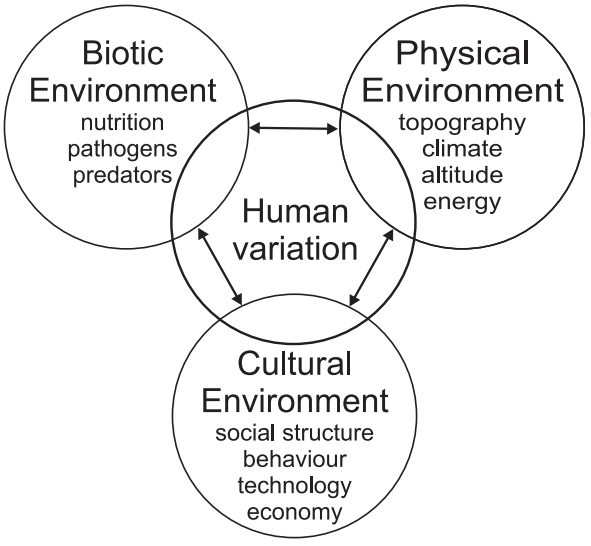



Fig. 6. Integrated biocultural model

praised at given point within this spectrum. The environmental insults that condition the health/disease process include both biologic and nonbiologic insults such as pathogens, toxins, physical forces, chemical pollutants, technology, and also social system, culture and ideology [Armelagos et al. 1992: 37]. Thereby, in any organism, health refers to the level of functional and/or metabolic efficiency of its hierarchic structure at both the micro (cellular, tissue and organ) and macro (environmental: biotic, physical, and societal) levels. Health means the ability of an organism to efficiently respond to environmental challenges (stressors) and effectively restore and maintain inputs and outputs of energy and matter in equilibrium, allowing for growth, development and reproduction and continuous survival.

Concepts of health/disease and a disease's perceived risk are broader than described above. According to Roberts, these concepts are shaped by cultural context, and are "…highly relevant to how a population understands the disease, how it is contracted and transmitted, and what measures may be put in place to combat the risk. These concepts have changed considerably over time as we have developed medical knowledge, especially about the pathogens that cause disease." (Roberts 2010: 22).

## The adolescent health study

*Research project* entitled: *At the doorstep to adulthood: adolescent health and quality of life in a variety of socio-economic backgrounds* (ADOPOLNOR) was implemented in 2008–2011 by the Adam Mickiewicz University in cooperation with Poznań University of Medical Sciences and Univeristy of Agder in Kristiansand, Norway. It was a multidisciplinary research project supported by the Research Funds 2008–2011 and co-financed by a grant from Iceland, Liechtenstein and Norway through the EEA Financial Mechanism and the Norwegian Financial Mechanism, under the *Academic Research* priority sector, contract number PL0255. All details of the project are available online at the website http://www.adopolnor.eu.

*The ethical and legal framework* The research proposal received approval from Bioethics Commission at Poznań University of Medical Sciences (Resolution no. 311/07).

Legal and ethical standards of the pediatric and anthropometric/physical fitness research that had been planned to be undertaken within school environment were also positively reviewed by Poznań Board of Education (this office handles all schools in the city of Poznań and the Wielkopolska province), and the consent was obtained to carry out research involving children and young people (Resolution WAF-405/1/JM/07).

With respect to the international and national governance frameworks, including the World Health Organization's *Declaration of Helsinki* on *Ethical Principles for Medical Research Involving Human Subjects*, the *Convention on the Right of the Child,* and *National Regulations on Ethics and Research in Poland*, a procedure of advising potential

participants and obtaining voluntary consent for ethically approved research project involving children and young people was applied.

Informed consent is a process by which a participant voluntarily confirms his or her willingness to participate in a particular piece of research, after having been informed fully of all aspects of the research that are relevant to the subject's decision to participate. Informed consent is documented by means of a written, signed and dated consent form. The consent process for research involving children and young people needs special consideration.

A child is defined by the Convention on the Rights of the Child (CRC) as "Every human being below the age of 18 years unless under the law applicable under the child majority is attained earlier". In Poland, full eligibility for legal actions is gained at the age of 18. Parents are the legal representatives for children and teenagers up to 18 years of age. The children, as appropriate for their developmental stage, provide assent. Assent is an individual's voluntary, affirmative agreement to participate in research. In Poland, if the minor is over 16 years of age or is under 16 years of age but is capable of expressing a conscious opinion about participation, his/her written consent is also necessary.

The informed consent process for the ADOPOLNOR research project began with the initial approach of an investigator to the potential study participants (including headmasters, teachers, nurses, parents and students) through information regarding the research study and continued until the completion of the research study. Information policy included face-to-face meetings with teachers, parents and students held in schools and the printed materials distributed among the target groups.

The leaflet regarding the research study contained following information: invitation to participate in the ADOPOLNOR research project; the purpose of the research and its main activities; that the research had been approved by Bioethics Commission at PUMS and Poznań Board of Education; the length of participation in the research and that participation is entirely voluntary; the right to withdraw from the research at any time without giving any reason; eligibility criteria before one can participate in the research; the possible benefits and risks of participation; about data storage and management and that data would be kept in confidence; contact details (including the website address) for further questions or request of withdrawal.

Almost all parents (97.1%) accomplished written informed consent for their children to participate in the ADOPOLNOR research project. Additionally, young people aged between 16 and 18 (96.7%) gave their written consent to be participants of the research.

*Aims and issues* The study aimed at drawing a comprehensive profile of physical growth and general health, subjective well-being and health-related quality of life of adolescents, aged 10–18 in the Wielkopolska region in relation to the socio-economic status of their families and their own health-related behaviour. The main premise of the study was that sociocultural context such as living standard conditioned by family's SES, lifestyle behaviours, and relationships with families, peer groups, school personnel, neighbourhood and community residents are important determinants of adolescent health and well-being at both individual and commu-

nity levels. Understanding this context is essential to effective and lasting modification of health-related behaviour.

The study addressed issues related to the status of adolescents' physical growth (with emphasis on height, which is strongly regulated by nutrition and health care), level of physical fitness, health-risk behaviour, quality of life expressed in terms of psychosocial well-being, chronic conditions and health care needs, and environmental determinants of health outcomes and well-being.

*Sampling frame for localities and schools in the study* The target population comprised adolescents aged 10–18, both gender students at elementary, junior secondary and senior secondary schools in the Wielkopolska province.

The sampling design employed in this study is basically a two-stage stratified cluster sampling plan in which (1) schools were stratified according to urbanization category thereby covering urban areas, including the city of Poznań, large and small towns, as well as rural areas, and randomly selected per each stratum. The development of the sampling frame comprised:

- developing a list of localities for Wielkopolska province broken down into: large cities (from 50,000 to 499,999), large towns of over 50,000, small towns below 49,999, and villages;
- developing a list of schools sorted by localities and types (elementary, junior secondary and senior secondary schools).

In the second stage of sampling procedure, (2) one class out of every six in the target population in each school was chosen. In most villages, however, the students were assigned to only one class of each year level group.

The ADOPOLNOR project was started in 2008, and the size of the sample was based on demographic statistics from 2007 (according to the Central Statistical Office). In 2007, the population of Wielkopolska province was 3,386,882 people; the urban population was 1,918,047 and rural was 1,914,841. Children and adolescents aged 10–18 accounted for 11.8% of the total population.

Given the administrative division of Poland, based on the NTS system (Nomenclature of Territorial Units for Statistics) applicable in the EU pursuant to the Regulation (CE) No.1059/2003 of European Parliament of 26 May 2003 (Official Journal EU L 154 from 21 June 2003), the country is divided into five levels:

- three regional levels including: regions, voivodeships (provinces), and subregions;
- two local levels including: poviats (districts) and communes.

The Wielkopolska province is divided in five subregions: Kalisz, Konin, Leszno, Piła, and Poznań. In 2007, the number of people living in particular subregions of Wielkopolska province was as follows:

- Kalisz – 667,734 people, and 20.6% of them accounting for population of pre-working age,
- Konin – 651,655 people, and 21.2% of them accounting for population of pre-working age,
- Leszno – 543,242 people; population of pre-working age accounted for 21.7% of the subregional population,

- Piła – 408,510 people; population of pre-working age accounted for 21.7% of the subregional population,
- Poznań – 569,212 people; population of pre-working age accounted for 21.5% of the subregional population.

Largest towns in subregions, in order from largest to smallest, are: Kalisz with population of 108,477 people; Konin – population of 80,471 people; Piła – population of 74,720 people; Ostrów – 74,492 people; Gniezno – 69,971 people, and Leszno – population of 63,955 people.

To establish a necessary young population to be covered with the study, it was assumed that the statistical power of tests assessing the difference between urban and rural areas had to be equal to 80%, and the level of significance at 5% ($\alpha = 0.05$).

The necessary size of the sample was established using the following formula [Brzeziński 1996: 247].

$$n = N/1 + d^2 (N-1)/u^2_{\lambda} \cdot pq$$

where:
n – necessary sample size
N – general population size
d – admissible estimation error of the p fraction
p – frequency of studied parameter, e.g. faulty posture, hypertension (1–3%)
u – normal distribution percentile $u_{\lambda} = z_{\lambda} = 1.64$ for $\lambda = 0.1$
$u_{\lambda} = z_{\lambda} = 1.96$ for $\lambda = 0.05$
$u_{\lambda} = z_{\lambda} = 2.58$ for $\lambda = 0.01$
$q = 1 - p$

The estimated necessary sample size was 4,460 individuals.

Localities selected for the survey included:

- the city of Poznań and other large cities such as: Piła, Konin, Kalisz,
- small towns: Turek, Zbąszyn, Sompolno, Krajenka, Bojanowo, Osieczna,
- villages: Biała, Głubczyn, Lubasz, Skórka, Opatówek, Lisków, Cisew, Słodków, Krzykosy (Pęczkowo, Sulęcin), Słupia Wielka.

Altogether, 52 schools of all levels were selected in the above listed localities.

Given the objectives of the study, it was assumed that the population of school children to be examined in Poznań was 1,206; in large and small towns 1,206 each; in rural areas 1,206 – total 4,836.

*Study design* This was a cross-sectional survey of almost 5,000 students conducted at 52 schools across the Wielkopolska province, including the city of Poznań, completed with a retrospective parents' account concerning physical growth since the moment of birth to a day of examination. The survey was carried out between February 2009 and September 2010. The protocol included face-to-face examinations such as pediatric screening, assessment of physical growth and physical fitness, taking biological samples for genetic tests and identification of herpes and human papilloma viruses, and interviews related to quality of life, including self-reported evaluation of body image. Students with chronic up-

per respiratory tract infections and with a high risk of cardiovascular diseases, obesity or high arterial pressure, underwent clinical tests and examinations.

*Study methodology* Aiming at assessing a general health status and disabilities, following tools were applied: the body measurements (somatometry), bioimpedance for assessing body composition, physical fitness tests, students' quality of life (YQOL) and body image (BIA) measured through self-report structured questionnaires, societal factors reported by parents and students in terms of SES and FAS questionnaires, molecular techniques.

Health disabilities, chronic conditions and health care needs were assessed using both external sources of epidemiological data for the province and survey data.

*Data analysis* Data base was created in Microsoft Excel 2007. All statistical computations were run using STATISTICA data analysis software system, version 9.0 [2009]. Analytical models comprised both uni- and multivariate approaches. The level of significance was set at $p<0.05$.

*Summary* The results of the study, presented in subsequent chapters of this volume, provided the researchers with current knowledge about adolescent physical health status (including physical growth and fitness) and quality of life, as well as risk and protective factors, encompassing individual, family, and extrafamilial (peer, school and community) factors.

Epidemiological data on risk and protective factors collected in the survey may be used to generate community risk profiles that will form the basis of recommendations for reducing prioritized risk factors and enhancing protective factors at a community level.

## References

Alsaker F.D.: Timing of puberty and reactions to pubertal changes. In: M. Rutter (Ed.) *Psychosocial Disturbances in Young People. Challenges for Prevention*. Cambridge, UK: Cambridge Univeristy Press 1995: 37–82.

Anderson C.A., Duffy D.L., Martin N.G., Visscher P.M.: Estimation of variance components for age at menarche in twin families. *Behav Genet* 2007; 37: 668–677.

Archer J.: Testosterone and human aggression: an evaluation of the challenge hypothesis. *Neurosci Biobehav R* 2006; 30: 319–345.

Arlt W., Martens J.W., Song M., Wang J.T., Auchus R.J., Miller W.L.: Molecular evolution of adrenarche: structural and functional analysis of p450c17 from four primate species. *Endocrinol* 2002; 143: 4665–4672.

Armelagos G.J., Leatherman T., Ryan M., Sibley L.: 1992. Biocultural synthesis in medical anthropology. *Med Anthropol 1*992; 14: 35–52.

Auchus R.J., Rainey W.E.: Adrenarche – physiology, biochemistry and human disease. *Clin Endocrinol* 2004; 60(3): 288–296.

Bertalanffy L. von: Principles and theory of growth. In: W.N. Nowinski (Ed.) *Fundamental Aspects of Normal and Malignant Growth* Amsterdam: Elsevier 1960: 137–259.

Bielicki T., Welon Z.: Growth data as indicators of social inequalities: the case of Poland. *Yearbook Phys Anthropol* 1982; 25: 153–167.

Bogin B.: *Patterns of human growth* (2nd ed.). Cambridge: Cambridge University Press, 2005.

Bogin B., Varela-Silva M. I.: Anthropometric variation and health: a biocultural model of human growth, *J Children's Health* 2003; 1(1): 149–172.
Brzeziński J.: *Metodologia badań psychologicznych*. Warszawa: PWN 1996.
Casey B.J., Jones R.M.: Neurobiology of the adolescent brain and behavior: implications for substance use disorders. *J Am Acad Child Psy* 2010; 49(12): 1189–1201.
Chunyan H., Kraft P., Chasman D.I., Buring J.E., Chen C., Hankinson S.E., Paré G., Chanock S., Ridker P.M., Hunter D.J.: A large-scale candidate-gene association study of age at menarche and age at natural menopause. *Hum Genet* 2010; 128(5): 515–527.
Coehlo A.M.: Baboon dimorphism: Growth in weight, length, and adiposity from birth to 8 years of age. In: E.S. Watts (Ed.) Nonhuman Primate Models for Human growth, New York: Alan R. Liss 1985: 125–159.
Colledge W.H.: GPR54 and puberty. *Trends Endocrinol Metab* 2004; 15: 448–453.
Conley A.J., Pattison J.C., Bird I.M.: Variations in adrenal androgen production among (nonhuman) primates. *Semin Reprod Med* 2004; 22(4): 311–326.
Delemarre-van de Waal H.A.: Regulation of puberty. *Best Pract Res Clin Obstet Gynaecol* 2002; 16: 1–12.
Del Giudice M., Angeleri R., Manera V.: The juvenile transition: A developmental switch point in human life history. *Dev Rev* 2009; 29: 1–31.
Dufour D.L.: Bio-cultural approaches in human biology. *Am J Hum Biol* 2006; 18: 1–9.
Ernst M., Fudge J.: A developmental neurobiological model of motivated behavior: anatomy, connectivity and ontogeny of the triadic nodes. *Neurosci Biobehav Rev* 2009; 33(3): 367–382.
Eveleth P.B., Tanner J.M.: *Worldwide Variation in Human Growth* 2nd ed. Cambridge University Press 1990.
Flinn M.V., Quinlan R.L., Ward C.V., Coe M.K.: Evolution of the human family: Cooperative males, long social childhoods, smart mothers, and extended kin networks. In: C. Salmon and T. Shackelford (Eds.) *Family Relationships*, Chapter 2, pp. 16–38. Oxford: Oxford University Press 2007.
Gluckman P.D., Hanson M.A.: Evolution, development and timing of puberty. *Trends Endocrinol Metab* 2006; 17: 7–12.
Golub M.S., Collman G.W., Foster P.M.D., Kimmel C.A., Rajpert-De Meyts E., Reiter E.O., Sharpe R.M., Skakkebaek N.E., Toppari J.: Public health implications of altered puberty timing. *Pediatrics* 2008; 121; S218-S230.
Grumbach M.M.: The neuroendocrinology of human puberty revisited. *Horm Res* 2002; 57(S2): 2–14.
Grumbach M.M.: Mutations in the synthesis and action of estrogen: the critical role in the male of estrogen on pubertal growth, skeletal maturation, and bone mass. *Ann N Y Acad Sci* 2004; 1038: 7–13.
Guo Y., Xiong D-H., Yang T-L., Guo Y-F., Recker R.R., Deng H-W.: Polymorphisms of estrogen-biosynthesis genes CYP17 and CYP19 may influence age at menarche: a genetic association study in Caucasian females. *Hum Mol Genet* 2006; 15: 2401–2408.
Halpern C.T., Udry J.R., Suchindran C.: Testosterone predicts initiation of coitus in adolescent females. *Psychosom Med* 1997; 59: 61–171.
Halpern C.T., Udry J.R., Suchindran C.: Monthly measures of salivary testosterone predict sexual activity in adolescent males. *Arch Sex Behav* 1998; 27: 445–465.
Hulanicka B., Kolasa E., Waliszko A.: *Dziewczęta z Górnego Śląska*. Monografie Zakładu Antropologii PAN, 1994, Wrocław.
Ibanez L., Dimartino-Nardi J., Potau N., et al.: Premature adrenarche, normal variant or forerunner of adult disease? *Endocr Rev* 2000; 21(6): 671–96.
James F.O., Cermakian N., Boivin D.B.: Circadian rythms of melatonin, cortisol, and clock gene expression during simulated night shift work. *Sleep* 2007; 30: 1427–1436.

Kaczmarek M.: *Poznańskie Badania Longitudinalne. Rozwój fizyczny chłopców i dziewcząt*. Monografie Instytutu Antropologii UAM 9. Poznań 2001.
Kaprio J., Rimpela A., Winter T., Viken R.J., Rimpela M., Rose R.J.: Common genetic influences on BMI and age at menarche. *Hum Biol* 1995; 67: 739–753.
Kotani M., Detheux M., Vandenbogaerde A., Communi D., Vanderwinden J.M., Le Poul E., Brezillon S., Tyldesley R., Suarez-Huerta N., Vandeput F. et al.: The metastasis suppressor gene KiSS-1 encodes kisspeptins, the natural ligands of the orphan G protein-coupled receptor GPR54. *J Biol Chem* 2001; 276: 34631–34636.
Kruger D.J., Wang X.T., Wilke A.: Towards the development of an evolutionary valid domain-specific risk-taking scale. *Evol Psychol* 2007; 5: 555–568.
Łaska-Mierzejewska T., Olszewska E.: The maturation rate of girls living in rich and poor rural regions of Poland before and after the transformation of 1989. *HOMO* 2004: 55 (1–2): 129–142.
Malina R.M., Bouchard C.: *Growth, Maturation, and Physical Activity*. Champagne, IL: Human Kinetics Press 1991.
Marshall W.A., Tanner J.M.: Variations in pattern of pubertal changes in girls. *Arch Dis Child* 1969; 44: 291–303.
Marshall W.A., Tanner J.M.: Variations in the pattern of pubertal changes in boys. *Arch Dis Child* 1970; 45: 13–23.
McElroy A.: Biocultural Models in Studies of Human Health and Adaptation. *Med Anthropol Quarterly* New Series Steps Toward an Integrative Medical Anthropology 1990; 4(3): 243–265.
Mitamura R., Yano K., Suzuki N., Ito Y., Makita Y., Okuno A.: Diurnal rhythms of luteinizing hormone, follicle-stimulating hormone, and testosterone secretion before the onset of male puberty. *J Clin Endocrinol Metab* 1999; 84: 29–37.
Muir A.I., Chamberlain L., Elshourbagy N.A., Michalovich D., Moore D.J., Calamari A., Szekeres P.G., Sarau H.M., Chambers J.K., Murdock P. et al.: AXOR12, a novel human G protein-coupled receptor, activated by the peptide KiSS-1. *J Biol Chem* 2001; 276: 28969–28975.
Northridge M.E., Elliott D. Sclar E.D., Biswas P.: Sorting out the connections between the built environment and health: a conceptual framework for navigating pathways and planning healthy cities. *J Urban Health* 2003 Dec; 80(4): 556–568.
Ohtaki T., Shintani Y., Honda S., Matsumoto H., Hori A., Kanehashi K., Terao Y., Kumano S., Takatsu Y., Masuda Y. et al.: Metastasis suppressor gene KiSS-1 encodes peptide ligand of a G protein-coupled receptor. *Nature* 2001; 411: 613–617.
Ojeda S.R., Lomniczi A., Sandau U., Martagne V.: New concepts on the control of the onset of puberty. In: S. Loche, M. Cappa, L. Ghizzoni, M. Maghnie, M.O. Savage (Eds.) *Pediatric Neuroendocrinology Endocr Dev* Basel, Karger, 2010; 17: 44–51.
Ong K.K., Potau N., Petry C.J., et al.: Opposing influences of prenatal and postnatal weight gain on adrenarche in normal boys and girls. *J Clin Endocrinol Metab* 2004; 89(6): 2647–2651.
Popma A., Vermeiren R., Geluk Ch.A.M.L., Rinne T., van den Brink W., Knol D.L., Jansen L.M.C., van Engeland H., Doreleijers T.A.H.: Cortisol moderates the relationship between testosterone and aggression in delinquent male adolescents. *Biol Psych* 2007; 61(3): 405–411.
Remer T.: Adrenarche and nutritional status. *J Ped Endocrinol Metab* 2000; 13(Suppl5): 1253–1255.
Reynolds M.D., Tarter R., Kirisci L., Kirillova G., Brown S., Clark D.B., Gavaler J.: Testosterone Levels and Sexual Maturation Predict Substance Use Disorders in Adolescent Boys: A Prospective Study. *Biol Psych* 2007; 61(11): 1223–1227.

Roa J., Navarro V.M., Tena-Sempere M.: Kissseptins in reproductive biology: consensus knowledge and recent development. *Biol Reprod* 2011 DOI:10.1095/biolreprod.111.091538.

Roberts Ch.: Understanding health: past and present. In: C. Panter-Brick and A. Fuentes (Eds.) *Health, Risk, and Adversity*. New York, Oxford: Berghan Books 2010.

Roemmich J.N., Clark P.A., Walter K., et al.: Physical activity energy expenditure, body composition, and abdominal fat distribution during puberty. *Am J Physiol Endocrinol Metab* 2000; E1426–1436.

Sisk C.L., Foster D.L.: The neural basis of puberty and adolescence. *Nat Neurosci* 2004; 7(10): 1040–1047.

Sisk C.L., Zehr J.L.: Pubertal hormones organize the adolescent brain and behavior *Front Neuroendo* 2005; 26: 163–174.

Somerville L.H., Casey B.J.: Developmental neurobiology of cognitive control and motivational systems. *Curr Opin Neurol* 2010; 20: 1–6.

Sørensen K., Aksglaede L., Petersen J., Juul A.: Recent changes in pubertal timing in healthy Danish boys: associations with body mass index. *J Clin Endocr Metab* 2010; 95(1): 263–270.

Spear L.P.: Adolescent brain development and animal models. *Ann N Y Acad Sci* 2004; 1021: 23–26.

Stavrou I., Zois C., Ioannidis J.P., Tsatsoulis A.: Association of polymorphisms of the oestrogen receptor a gene with the age of menarche. *Hum Reprod* 2002; 17: 1101–1105.

Stavrou I., Zois C., Chatzikyriakidou A., Georgiou I., Tsatsoulis A.: Combined estrogen receptor $\alpha$ and estrogen receptor $\beta$ genotypes influence the age of menarche. *Hum Reprod* 2006; 21: 554–557.

Steinberg L., Belsky J.: A sociobiological perspective on psychopathology in adolescence. In: D. Cicchetti and S. Toth (Eds.) Rochester Symposium on Developmental Psychopathology. University of Rochester Press; Rochester, NY: 1996: 93–124.

Stinson S., Bogin B., Huss-Ashmore R., O'Rourke D.: Human Biology: An Evolutionary and Biocultural Perspective. New York: Willey-Liss 2000.

Styne D.M., Grumbach M.M.: Control puberty in humans. In: O.H. Pescovitz and E.C. Walvoord (Eds.) *When Puberty is Precocious: Scientific and Clinical Aspects*. Totowa, NJ: Human Press Inc. 2007; 51–67.

Susman E.J., Inoff-Germain G., Nottelmann E.D., Loriaux, D.L. Cutler Jr. G.B., Chrousos G.P.: Hormones, emotional dispositions, and aggressive attributes in young adolescents. *Child Dev* 1987; 58: 1114–1134.

Szafraniec K. (Ed.) *Młodzi 2011* available at http://www.zds.kprm.gov.pl/node/185. Accessed 29 August 2011.

Tanner J.M.: *Growth and Adolescence*. 2nd ed. Oxford: Blackwell Scientific Publications; 1962.

Tanner J.M.: *Growth* (Life Science Library). Time-Life Books, Alexandria, USA; Revised edition. 1981.

Tanner J.M.: Hormonal, genetic, and environmental factors controlling growth. In: G.A. Harrison, J.M. Tanner, D.R. Pilbeam, P.T. Baker (Eds.) *Human Biology. An Introduction to human Evolution, Variation, Growth, and Adaptability*. 3rd ed. Oxford, New York, Tokyo: Oxford University Press; 1989: 377–395.

Tanner J.M., Whitehouse R.H., Marshall W.A., Carter B.S.: Prediction of adult height, bone age, and occurrence of menarche, at ages 4 to 16 with allowance for midparental height. *Arch Dis Child* 1975; 50: 14–26.

Tena-Sempere M.: GPR54 and kisspeptin in reproduction. *Hum Reprod Update* 2006; 12(5): 631–639.

Tena-Sempere M., Barreiro M.L.: Leptin in male reproduction: the testis paradigm. *Mol Cell Endocrinol* 2002; 189: 9–13.

Tena-Sempere M., Huhtaniemi I.: Gonadotropins and gonadotropin receptors. In: B.C.M.J. Fauser (Ed.) *Reproductive Medicine – Molecular, Cellular and Genetic Fundamentals*. New York: Parthenon Publishing 2003: 225–244.

Terasawa E., Fernandez D.L.: Neurobiological mechanisms of the onset of puberty in primates. *Endocr Rev* 2001; 22: 111–151.

Terasawa E., Kurian J.R., Guerriero K.A., Kenealy B.P., Hutz E.D., Keen K.L.: Recent discoveries on the control of GnRH neurons in nonhuman primates. *J Neuroendocrinol* 2010; 22(7):630–638. doi:10.1111/j.1365-2826.2010.02027.x.

Towne B., Czerwinski S.A., Demerath E.W., Blangero J., Roche A.F., Siervogel R.M.: Heritability of age at menarche in girls from the Fels Longitudinal Study. *Am J Phys Anthropol* 2005; 128: 210–219.

Veldhuis J.D.: Neuroendocrine facets of human puberty. *Neurobiol Aging* 2003;24:S93-S119.

*WHO 10 facts on adolescent health September 2008* Available at: http://www.who.int/features/factfiles/adolescent_health/facts/en/index.html, Accessed February 10, 2011.

Wolański N.: *Biologiczne podstawy rozowju człowieka. Podstawy auksologii, gerontologii i promocji zdrowia* Wydanie: siódme, zmienione. Wydawnictwo Naukowe PWN 2006.

Wolański N.: *Ekologia człowieka. Podstawy ochrony środowiska i zdrowia człowieka. T. 1, Wrażliwość na czynniki środowiska i biologiczne zmiany przystosowawcze* Wydawnictwo Naukowe PWN 2008.

Wu F.C., Butler G.E., Kelnar C.J., Huhtaniemi I., Veldhuis J.D.: Ontogeny of pulsatile gonadotropin releasing hormone secretion from midchildhood, through puberty, to adulthood in the human male: a study using deconvolution analysis and an ultrasensitive immunofluorometric assay. *J Clin Endocrinol Metab* 1996; 81: 1798 –1805.

# Health and Enviroment

Maria Kaczmarek, Magdalena Skrzypczak

# Health-related factors of natural and socio-economic environments in Wielkopolska province, 2005–2009

**Abstract:** This paper presents built/physical, social, economic, and natural environmental dimensions of health resources in Wielkopolska province in the years 2005–2009. Using 19 indicators of environmental health, including bioclimate, environmental assets and degree of environment degradation, indicators of demographic situation, living standard and the wealth of the population, an overview of territorial diversity in natural and socio-economic health factors affecting people in Wielkopolska province was accomplished. Research findings revealed that urban areas were favourable for human health in terms of societal factors and disadvantageous due to bio-geographic conditions. In contrary, rural areas were less permissive for social and economic living conditions but favourable for natural and physical environments. The notion of environmental health resources is important for successful promotion of health and well-being.

**Key words:** environmental health, commune clusters, health promotion

## Introduction

Human health is a multidimensional concept defining physical, mental and emotional status of an individual and a community. Health viewed from an individual perspective is inscribed into a life cycle as an outcome of the interaction that occurs between human genotype and the external environment. Social health implies building and maintaining good interpersonal relations; it pertains to wellbeing of an individual and a community. Health factors, depending on their origin, scope and extent of action, have come to be classified into the categories of human biology, lifestyle, environment and healthcare organisation [Lalonde 1974].

Factors relating to human biology comprise the genetic make-up that forms a matrix for phenotypic traits developing in interaction with the environment, genetic predisposition to diseases, deterioration of biological status and age-related loss of physical and mental health capacity, specific lifestyle, stress, injuries or infections. It also includes a family history of diseases.

Environment for life embraces all non-hereditary components of human ontogenetic environment that regulate the course of development processes through general activities, such as the impact of natural factors or nutritional habits, or specific activities (local pH, $O_2$ concentration or osmotic balance). Human living environment is affected by natural conditions, such as topography, water supply, climate or wildlife, and socio-economic factors which are produced by individual or collective actions of community members. Socio-economic factors act on human health through the living and work conditions, social relationships and welfare, social standards, culture and religion. They are characteristic for a given society, reflecting its culture and established social standards. Biodiversity, sufficient food supply, clean air and water, UV protection, waste disposal and recycling are all favourable health contributors. On the other hand, health can be threatened by invasive biota (bacteria, viruses), natural disasters (floods, hurricanes, fires, earthquakes), man-induced calamities (famine, violence, war) or environmental transformations caused by human activity (urbanisation, industrialisation, air and water pollution).

Lifestyle is about the way a person or group of people live, his/her, their health-promoting or health-threatening behaviour, standards, habits, beliefs and stress management abilities [Anderson 1984; Edgar, Sedgwick 1999]. The definition of lifestyle encompasses more aspects of daily living than most people would realize. Following facets that make up a lifestyle have usually been pointed out: habits and lifestyle family fitness (everything a person does on a habitual or regular basis), the career or employment opportunities a person pursues, financial means and emotional well-being.

A healthcare system in a country is another important category of health factors involving organisation, financial resources, and scope, availability and quality of medical service. The system is aimed to promote health and prevent diseases.

The above listed factors work comprehensively being tied with one another by a network of interdependencies. Given the complexity of the system, it is difficult to assess the impact of any particular factor, although we realise each of them contributes to health status in a different extent [Wilson et al. 1998]. This knowledge however allows to distinguish areas of specific morbidity risk levels [Mitchell, Popham 2008; Poniży 2008].

One of those areas is environmental health, a category of public health. based on the understanding of how the natural environment and socio-economic conditions affect the health status of a population [WHO 2007; World Bank 2008; Marsili 2009].

"Environmental health addresses all the physical, chemical, and biological factors external to a person, and all the related factors impacting behaviours. It encompasses the assessment and control of those environmental factors that can potentially affect health. It is targeted towards preventing disease and creating health-supportive environments. This definition excludes behaviour not related to environment, as well as behaviour related to the social and cultural environment, and genetics" [http://www.who.int/topics/environmental_health/en/Accessed March 1, 2011].

Research into environmental aspects of medical geography has shown a relationship between selected types of bioclimate and health [Kozłowska-Szczęsna, Krawczyk 1990. Kozłowska-Szczęsna et al. 1997. Malinowska 2002]. A correlation has also been found between environmental degradation in a city and the health status of a population [Poniży 2008]. Michalski [2002, 2002a] demonstrated a differentiating impact a place of residence has on health. Chojnicki and Czyż introduced the concept of urban-rural continuum to describing the urbanisation gradient of human health [Chojnicki. Czyż 1989].

A commune, district or province with degraded living environments, low-income levels, high morbidity and mortality rates, low quality of life, and insufficient or poor quality healthcare systems will fail to attract newcomers or investors looking for natural or human resources. thus perpetuating or deepening the existing state of affairs [Michalski 2002a:45].

The impact of urbanization factor and socio-economic status of the family on human physical and mental development is a phenomenon widely recognized by human auxologists [Bielicki, Welon 1982; Danker-Hopfe 1986; Tanner 1986; Bielicki, Waliszko 1991; Rona 2000].

Results of the above cited studies show that the incorporation of environmental health issues into spatial planning policies of territorial units is essential in increasing their chances for sustainable development.

The objective of this study is to describe environmental health conditions of Wielkopolska population and indicate territorial units that are characterised by higher risk of morbidity because of those conditions.

## Research methodology – indicators of the natural and social environment quality

WHO, UN and the European Commission recommend a set of indicators to assess the quality of human living environment. With regard to the socio-economic environment, the Core Health Data System comprises indicators concerning demography, socio-economic status, mortality, morbidity and risk factors, resources, services and welfare. The quality of natural environment is in turn determined by indicators of air quality, water quality and build environment not mention climate changes[www.who.eu; un.gov.eu; eu.gov.com].

However, the literature of the subject fails to provide a clear-cut framework of indicators defining natural and socio-economical conditions of human health, which seemed to have been a major methodological setback in developing this study. Therefore, in selecting the indicators, we relied on the availability of relevant statistics and comparability of obtained results with earlier publications [Mazurkiewicz, Wróbel 1990; Kozłowska-Szczęsna et al. 1997; Michalski 2000, 2000a; Malinowska 2002; Kozłowska-Szczęsna et al. 2004; Poniży 2008].

We found climate, in particular temperature, humidity, atmospheric pressure, air movements and insolation, land relief, type of soil, water supply, wildlife, as

well as parasites, bacteria and viruses as factors of relevance for human living environment, which can undergo degradation through human use and thus lose its natural assets.

Population density, distribution of gender, age and education level, birth rate, life expectancy, housing standards; social infrastructure; and healthcare organisation. all add up to constitute a social environment of human life. Technical infrastructure, unemployment rate and income level go together to make up a standard of living. Socio-economic living conditions are related to a specific lifestyle. Unemployment, poverty, low education level, and poor housing conditions may encourage risky behaviour and social pathologies and in consequence adversely affect health status.

To describe health-related environmental factors we chose to make use of indicators pertaining to 1) bioclimate 2) environmental assets and 3) degree of environment degradation.

The concept of bioclimate was introduced by Humboldt in 1827, although the relationship between weather conditions and human physical and mental well-being had been known long before. Humboldt defined bioclimate as "a complex of atmospheric factors that affect human sensual receptors" [Kozłowska-Szczęsna et al. 1997:17]. The currently applied definition of extends the scope proposed by Humboldt to non-specific, systemic impact of climatic factors, and the influence of electromagnetic radiation of various frequency and ionising radiation. The new approach proposes to evaluate climatic conditions in the context of their biological effect on humans and other living organisms [Parsons 2003].

This study refers to the types of bioclimate as distinguished of Poland in research works on the intensity of influence particular atmospheric stimuli have on human organism [Kozłowska-Szczęsna et al. 1997:168–172]. The following abiotic stressors were identified: 1) mean annual number of freezing days (temperature below –10°C), 2) mean annual number of hot days (maximum temperature over 30°C), 3) mean annual number of sultry days (human organism perceives such conditions as oppressive) and 4) mean annual number of stormy days.

Environmental assets are described by the following indicators: 1) share of forest area in the total land area; 2) proportion of green area in the total land area and 3) proportion of legally protected areas in the total land area. The following indicators were used as determinants of environment pollution: 1) amount of waste collected in a commune in tonnes per year; 2) particulate emission levels from major polluting plants in tonnes per year (data for 2005 and 2009); 3) gas emission levels from major polluting plants in tonnes per year (data 2005 and 2009); 4) distribution of municipal and industrial landfills in the province of Wielkopolska and 5) distribution of industrial emitters in the province of Wielkopolska (industrial plants emitting particulates or gases to the atmosphere).

The following indicators were used to determine demographic situation of the region: 1) population density; 2) feminisation ratio; 3) percentage of post-productive population to total population; 4) rate of live births per 1,000 population; 5) rate of deaths per 1,000 population; 6) rate of infant deaths per 1,000 population; 7) rate of natural increase per 1,000 population; 8) rate of net migration per 100 population and 9) life expectancy at birth.

Living conditions of Wielkopolska population were determined basing on the following indicators: 1) average usable floor space of dwellings per person in $m^2$; 2) dwellings completed per 1,000 population; 3) percentage of population using water-supply system (in % of total population); 4) percentage of population using sewerage system (in % of total population).

The wealth level of a society is measured in relation to Gross Domestic Product per capita and average monthly remuneration. Both not being available at a commune level, the wealth level of a commune is for the purposes of this study derived from its revenue from personal and corporate income tax. The proportion of registered unemployed persons to the total productive population and the level of communal expenditure on housing benefits were used as indicators of poverty.

The spatial scope of the study covered the province of Wielkopolska. The basic unit of the analysis is a commune subdivided into rural, urban-rural, and rural communes. Additionally the territory of Wielkopolska province was divided into urban areas, comprising urban communes, city districts and urban parts of urban-rural communes, and rural areas. encompassing rural communes and rural parts of urban-rural communes. Due to insufficient data. certain analyses were limited to subregions or the province as a whole. The study covers the period of 2005–2009.

The classification of Wielkopolska communes in terms of natural and socio-economic health factors was made by means of the cluster analysis using the *k-mean* method [Chojnicki, Czyż 1973; Parysek 1982]. The method involves the division of the whole set into *k-groups,* so as to minimise the intra-group variation, using Euclid's distance between objects as a measure of similarity. First, a classification was developed through uni-dimensional analyses for each of the studied indicators separately and for the period of 2005–2009. Their results are presented in tables and charts. Then, a multi-variable analysis was made. The investigation employed a selected set of 19 above-listed indicators specific for health conditions in the communes of Wielkopolska province in 2007 and 2009. It was assumed that the *k-mean* method and 20 iterations would be applied to distinguish 5 principal classes of communes. This way, we were able to generate an overview of territorial diversity of natural and socio-economic health factors affecting Wielkopolska population.

## Results

### Natural health factors in Wielkopolska province

The borders of Wielkopolska province were set in 1999 following the reform of Poland's territorial administration. The region covers the area of 29,826 sq. kilometres, accounting for 9.5% of the total area of Poland and making the second largest province behind Mazovia.

Table 1 shows selected data on the natural and social environment of Wielkopolska province. Those data were compared with those concerning the whole country to illustrate the specificity of the region

Table 1. Selected data on natural and social environment of Wielkopolska and Poland[a]

| Specification | Poland | Wielkopolska |
|---|---|---|
| **Climate** | | |
| Average air temperature in °C | 7 | 9.1 |
| Total annual atmospheric precipitation in mm | 683 | 584 |
| Insolation in h | Warsaw 2258 | Poznań 1852 |
| Average wind speed in m/s | Warsaw 3.0 | Poznań 3.5 |
| **Natural environmental assets** | | |
| Forests in thous. ha | 9088 | 763.2 |
| Legally protected areas with unique environmental value in % of total area | 32.3 | 31.8 |
| **Devastated and degraded land** | | |
| requiring reclamation and managements in ha | 62,077 | 10,009 |
| Emission of industrial air pollutants in thous.t | | |
| particulates | 61.7 | 8.4 |
| gases | 1590.7 | 78.8 |
| Waste generated during the year | | |
| per 1 km of total area in t | 355 | 190 |
| treated by storage in % of total | 19.2 | 22.7 |
| **Built environment** | | |
| Total area in $km^2$ (in percent) | 31,2679 (100) | 29,826 (9.5) |
| Land use: | | |
| Agricultural land in thous. ha (in percent) | 16,112 (100) | 1,807 (11.2) |
| Built up and urbanized areas in thous. ha | 1550 | |
| Transportation systems | | |
| Railway lines operated (standard gauge) in km | 20,171 | 2022 |
| Hard surface public road per 100 $km^2$ | 83.6[b] | 87.9[b] |
| Services | | |
| Beds in general hospitals per 10 thous. population[b] | 48.1 | 47.2 |
| Consultations provided within the scope of outpatient health care per capita | 7.5 | 7.1 |
| Children in nurseries and nursery wards per 100 children up to age 3 | 25.8 | 18.7 |
| Audience in cinemas per 1000 population | 1024 | 1004 |
| Subscribers per 1000 population | | |
| radio | 184 | 202 |
| television | 178 | 195 |

| Specification | Poland | Wielkopolska |
|---|---|---|
| **Demography** | | |
| Population in thous. (in percent) | 38,167.3 (100) | 3,408.3 (8.9) |
| Females per 100 males | 107 | 106 |
| Infant deaths per 1,000 live births | 5.6 | 5.4 |
| Population growth per 1,000 population | 11.9 | 9.1 |
| **Socio-economic status** | | |
| Average monthly gross remuneration in PLN | 3,315 | 2,976 |
| Dwellings per 1,000 population | 349 | 320 |
| Usable floor space of dwellings per person | 24.6 | 24.9 |
| Registered unemployment rate in % | 11.9 | 9.1 |
| GDP in 2007 per capita in PLN (current prices) | 30,873 | 32,266 |

Explanations: [a]source: Central Statistical Office data for 2009; available at www.stat.gov.pl, accessed: February 24, 2011

Geographically, Wielkopolska is located within the boundaries of the Central Polish Lowlands and the South Baltic District Area. The southern part of the province was modelled by the Riss glaciation, while the northern part was formed in the last glacial period. The activity of the ice sheet caused the terrain of Wielkopolska to be flat and rolling with numerous hills and lakes mainly in the northern part. The whole region is situated in the drain basin of the Oder river [Kondracki 1998].

In terms of bioclimate, the area offers favourable conditions for human life, being weakly stimulating with less than 20% of days with oppressive weather conditions during a year. Only in the bigger cities of Poznań, Konin, Kalisz, Leszno and Piła and the industrial zones (lignite and gas mining) is the bioclimate less favourable as is typical for highly urbanised areas. Large woodlands, such as Noteć Forest and Zielonka Forest, exhibit features of conservative bioclimate [Kozłowska-Szczęsna et al. 1997].

Wielkopolska province lies in the moderate climate zone. Climatic conditions do not show a high degree of spatial diversification. The mean annual air temperature is 9.1°C. In the northern part of Wielkopolska, the mean annual temperature is 8.6°C, growing south-westwards up to 9.6°C. In the warmest month of July, the mean temperature is 18°C, while in the coldest month of January it falls down to –1°C. Winters tend to be short and mild. The mean annual precipitation is lower than Poland's average amounting to 584 mm. Only in the northern and southern parts of the province does the total annual precipitation exceed 600 mm. The mean wind speed ranges between 3 to 3.5 m/s, and relative humidity is 80%. The mean annual atmospheric pressure is in the range of 1015–1017 hPa.

The mean annual number of freezing days is below 2 and grows eastwards. The mean annual number of hot days is 8. The most sultry days occur in the west of the province up to 24 with less than 20 in the north. The mean annual number of days with thunderstorms is 20 [Farat ed. 2004: 56, 58, 114 and 119].

Table 2 shows indicators for evaluation of natural environment assets in the province of Wielkopolska 2005–2009. Further figures present spatial variation of those indicators using a five-level scale. The figures refer to the years 2005–2009.

Table 2. Natural environmental assets in Wielkopolska province by commune types in the period 2005–2009 years

| Specification | Type of commune | 2005 | 2006 | 2007 | 2008 | 2009 |
|---|---|---|---|---|---|---|
| Forest area (in %) | total | 25.03 | 25.53 | 25.55 | 25.55 | 25.29 |
| proportion of commune | urban | 16.21 | 16.22 | 16.19 | 16.29 | 16.33 |
| area (in %) | urban-rural | 27.34 | 27.38 | 27.40 | 27.40 | 27.44 |
| | rural | 24.12 | 24.12 | 24.15 | 24.15 | 24.18 |
| | urban areas | 14.52 | 14.54 | 14.50 | 14.53 | 14.54 |
| | rural areas | 26.08 | 26.10 | 26.13 | 26.13 | 26.17 |
| Green areas (in %) | total | 0.10 | 0.10 | 0.10 | 0.10 | 0.10 |
| proportion of commune | urban | 1.22 | 1.10 | 0.91 | 0.90 | 0.89 |
| area (in %) | urban-rural | 0.08 | 0.08 | 0.08 | 0.08 | 0.08 |
| | rural | 0.05 | 0.06 | 0.06 | 0.06 | 0.06 |
| | urban areas | 1.10 | 1.02 | 0.92 | 0.91 | 0.90 |
| | rural areas | 0.05 | 0.05 | 0.05 | 0.05 | 0.05 |
| Legally protected | | | | | | |
| areas (in %) | total | 31.01 | 31.40 | 31.55 | 31.57 | 31.79 |
| proportion of commune | urban | 7.92 | 7.91 | 8.64 | 8.88 | 10.73 |
| area (in %) | urban-rural | 31.01 | 31.43 | 31.14 | 31.15 | 31.67 |
| | rural | 32.29 | 32.68 | 33.26 | 33.28 | 33.10 |
| | urban areas | 4.29 | 4.29 | 4.68 | 4.70 | 4.71 |
| | rural areas | 31.26 | 31.46 | 31.75 | 31.76 | 31.78 |

The proportion of forest area to the total area of the province remained unchanged throughout the studied period averaging 25.5%. The ratio did not exceed 28% in the urban-rural communes and 14.5% in towns and cities. In respect of forest cover, a note should be taken of the northern and southern part of Wielkopolska (Fig. 1). Forests occupy over 30% of the total land area of those regions. The equivalent ratio is below 10% for urban and rural areas located in the central and eastern part of the province.

As regards green areas, only in urban communes and cities comprised by the urban-rural communes did they take up more than 1% of the total land area. Communes of that type are mostly situated in the central part of the province (Fig. 2). The absence or meager proportion of greens was found to be typical for northern Wielkopolska where forests occupy much of the area. In general, the ratio of green area per total land area of the region is very low, reaching no more than 0.1%.

More than 30% of rural and urban-rural communes (mainly rural parts thereof) were covered by various forms of legally protected areas, such as national parks, nature reserves and landscape parks. The proportion of such areas in urban communes was below 9%. Communes with high share of protected areas are mainly to be found in the south (including "Dolina Barycz" Landscape Park), centre (most notably Wielkopolska National Park), north-west (Drawa National Park, Krajenka

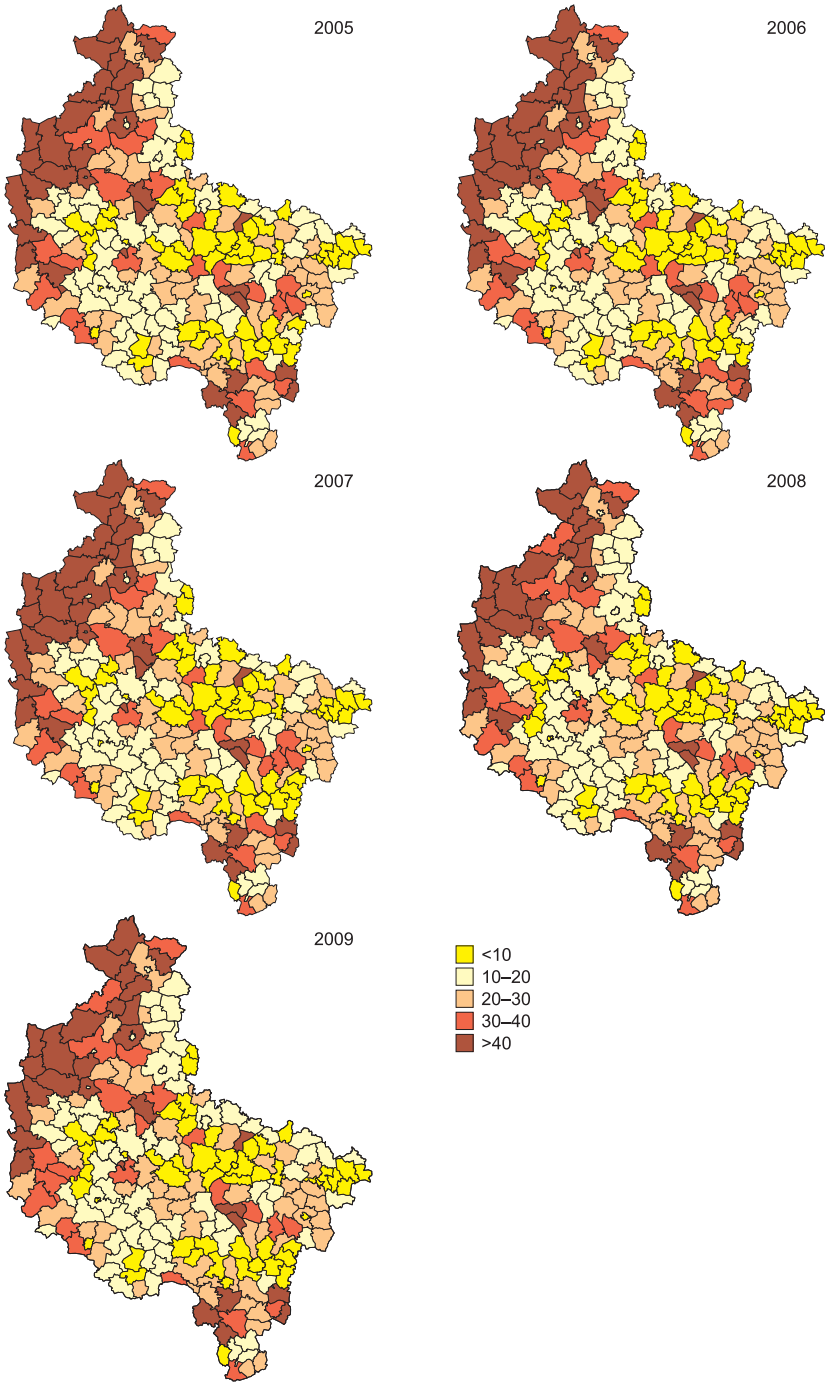



Fig. 1. Forest areas in communes of Wielkopolska province in 2005–2009 by commune types (as percentage of commune area)

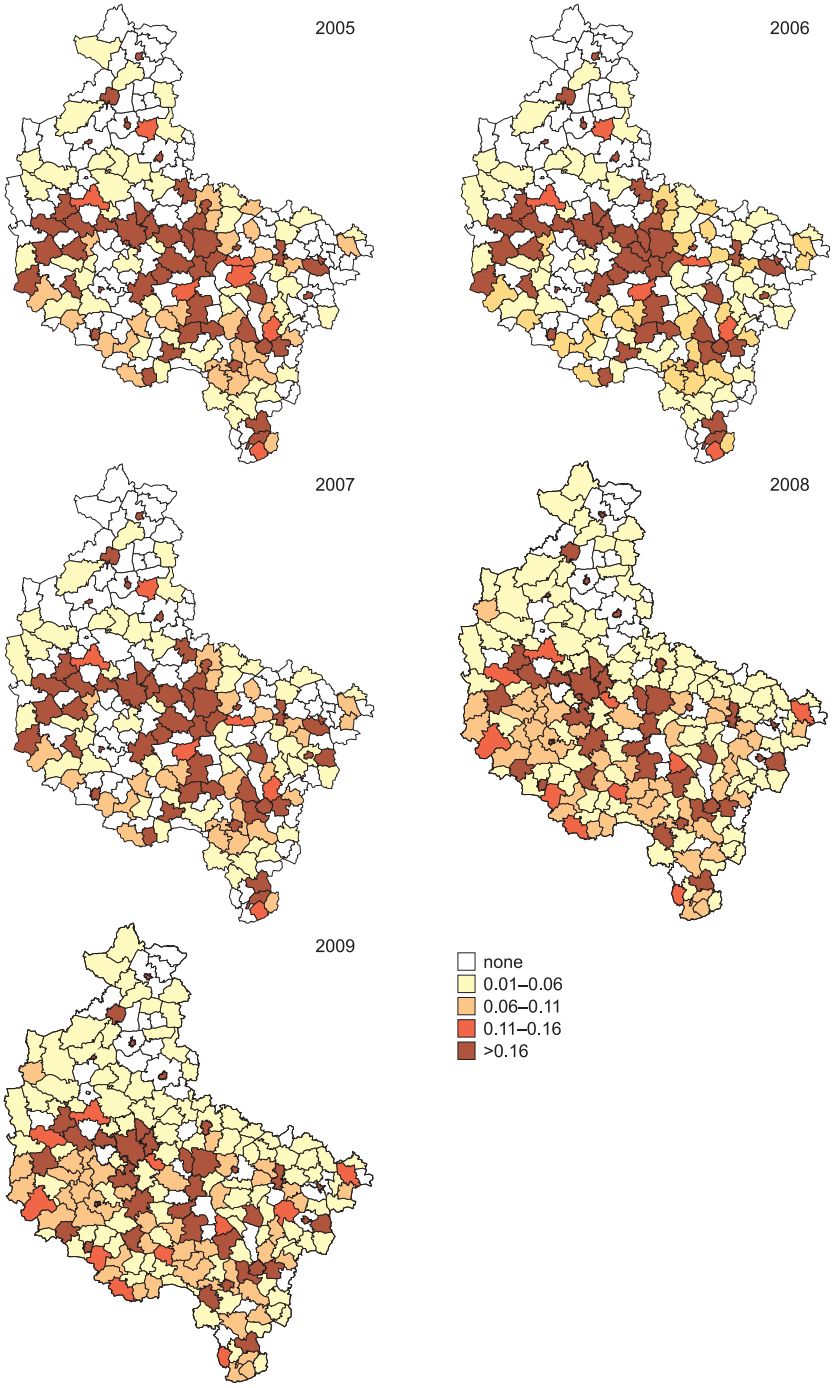



Fig. 2. Green areas in communes of Wielkopolska province in 2005–2009 by commune types (as percentage of commune area)

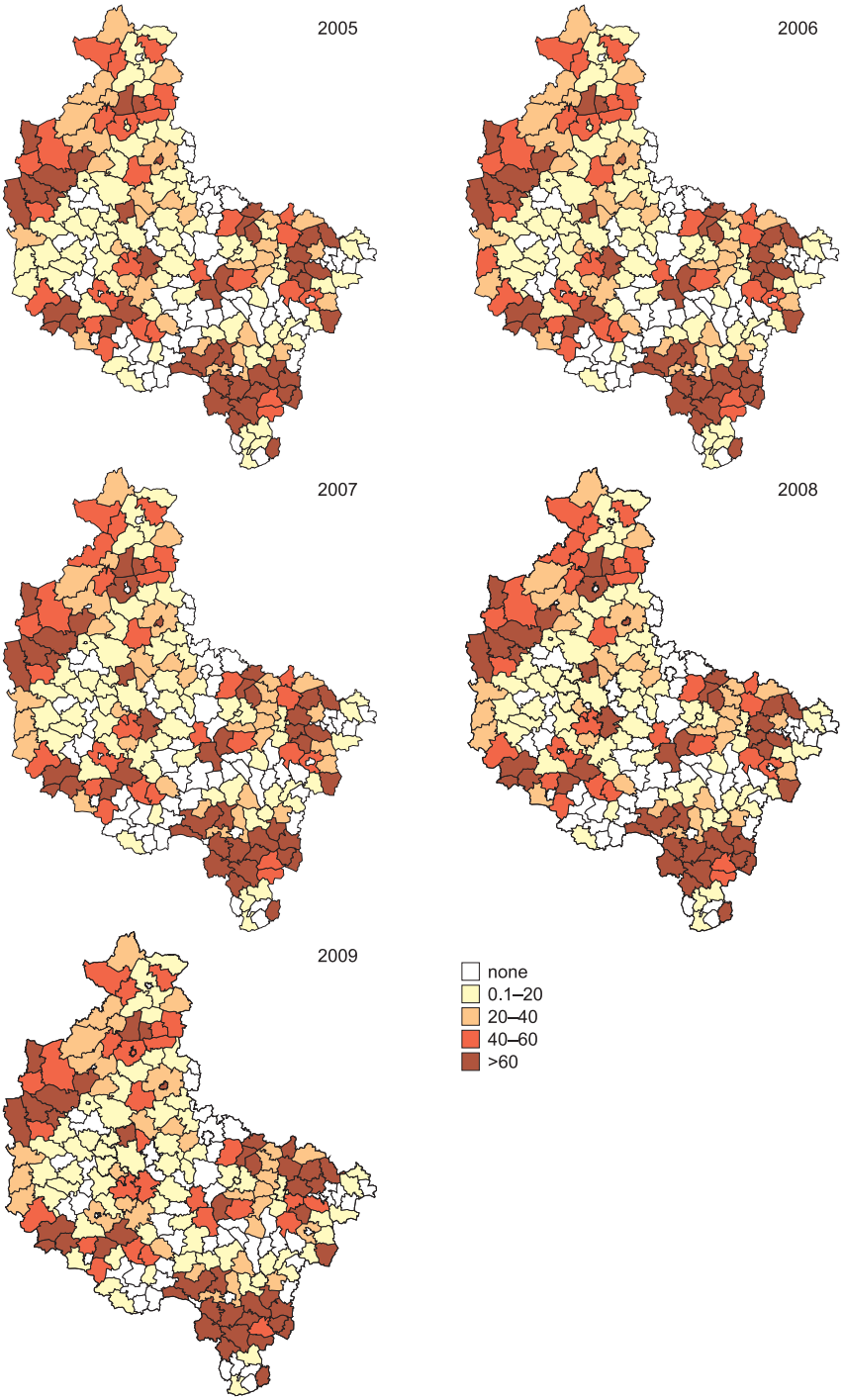



Fig. 3. Legally protected areas with unique environmental value in percent of total area

Table 3. Environment degradation indices in Wielkopolska province, 2005–2009

| Specification | | 2005 | 2006 | 2007 | 2008 | 2009 |
|---|---|---|---|---|---|---|
| Waste generated during the year (in thousands of tonnes) | | 832.1 | 848.2 | 809.6 | | |
| Emission of air pollutants during the year (in thousands of tonnes) | | | | | | |
| particulates | | 9.7 | 10.3 | 7.0 | 6.6 | 6.6 |
| gases | | 16,987.3 | 17,975.6 | 18,294.2 | 18,264.7 | 17,595.6 |
| Specification | Type of commune | 2005 | 2006 | 2007 | 2008 | 2009 |
| Emission of air pollutants during the year (in thousands of tonnes) | total | 16,987.3 | 17,975.6 | 18,294.2 | 18,264.7 | 17,595.6 |
| gases | urban | 16,132.8 | | | | |
| | urban-rural | 769.6 | | | | |
| | rural | 84.9 | | | | |
| | urban areas | 16,853.1 | | | | |
| | rural areas | 134.2 | | | | |

Landscape Park) and east (Powidz Landscape Park, Warta Landscape) of the region. Distribution of legally protected areas with unique environmental value in % of total area is shown in Figure 3.

Table 3 shows indicators for evaluation of natural environment degradation in the region of Wielkopolska in the period 2005–2009. Urban communes are the biggest polluters with over 1.3 million tonnes of waste produced annually. Large amounts of waste are also generated by urban-rural and rural communes situated in the middle part of Wielkopolska province, mainly in the Poznań district (Fig. 4). The cleanest communes, with less than 1,000 tonnes of waste collected are those in the south-east of the region. These are mainly rural communes.

Collected waste is directed to municipal or industrial landfills. The communes of central Wielkopolska, that is those producing the largest amounts of waste, have relatively fewer landfills than those in other parts (Fig. 5). The largest number of landfills is situated in the southern and eastern part of the province.

Spatial distribution of industrial emitters and particulate and gas emissions from heavy polluting plants in Wielkopolska region indicates a large degree of concentration (Fig. 6) The largest number of emitters and the highest extent of emission are recorded in highly urbanised centres, such as Poznań, Konin, Kalisz, Piła, Leszno, Ostrów Wielkopolski as well as the areas holding mining and energy industries (marked light and dark red in the Figure). The communes of eastern Wielkopolska are characterised not only by lower number of industrial polluters but also by lower levels of particulate and gas emissions (less than 1 tonne per year).

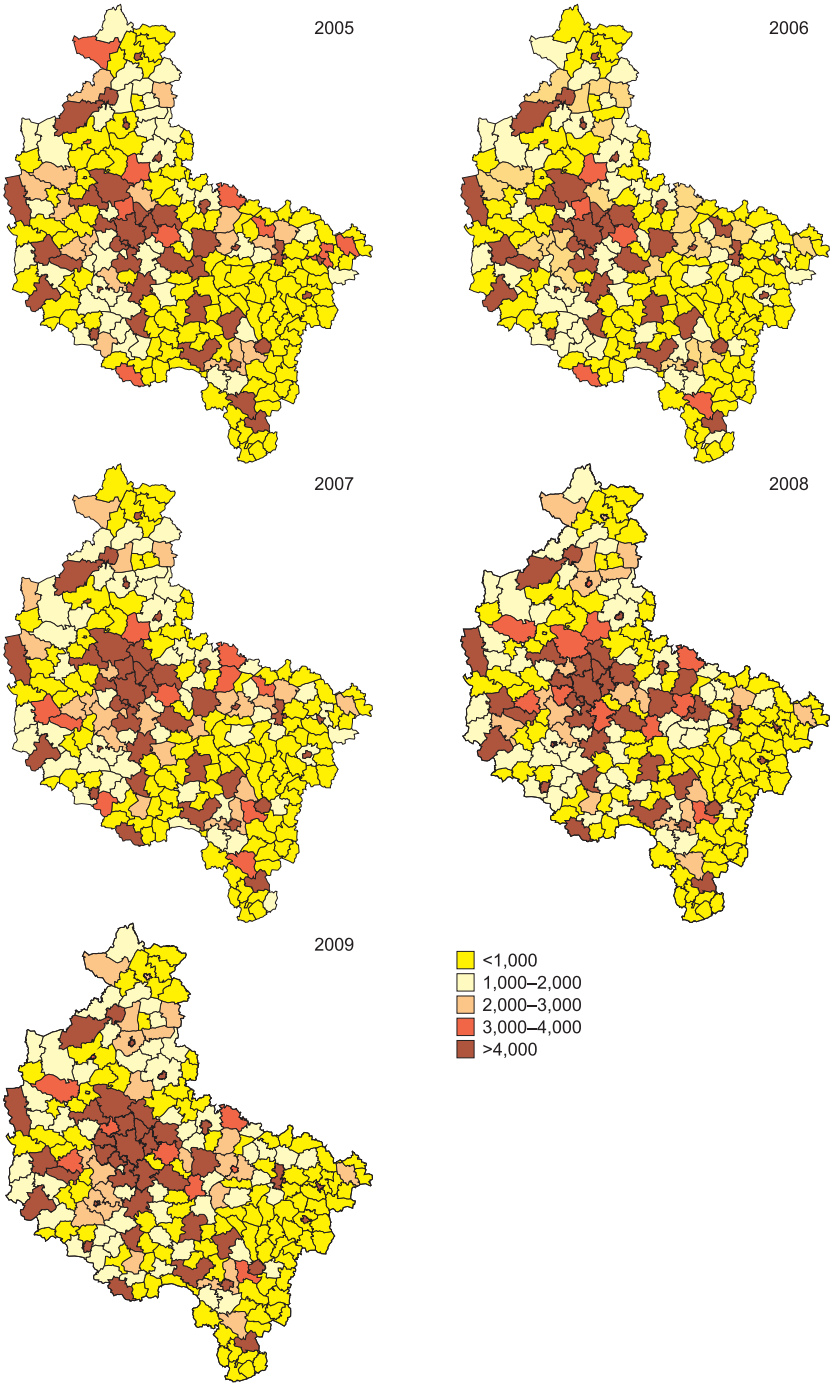



Fig. 4. Waste collected in communes of Wielkopolska province, 2005–2009 (tonnes per years)

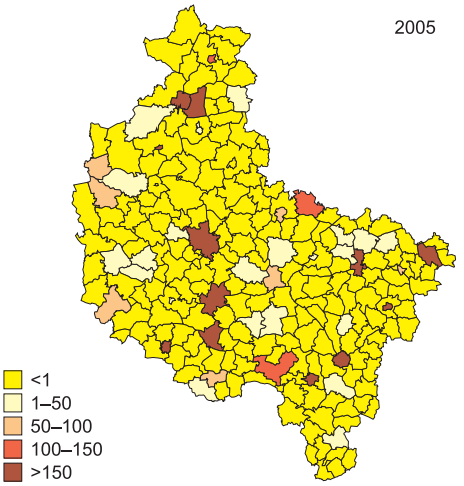



Fig. 5. Landfills and industrial emitters in communes of Wielkopolska province in 2005, sozological map 1:50,000

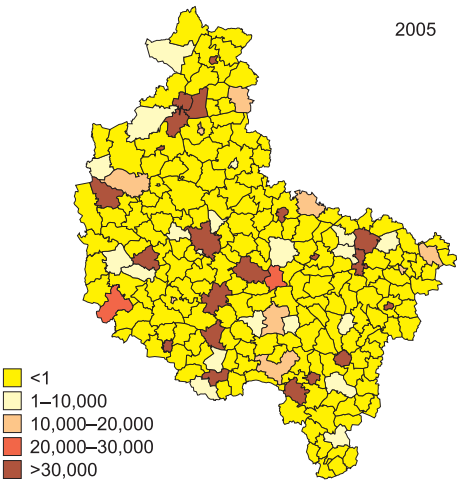



Fig. 6. Particulate and gas emissions from heavy polluting plants in Wielkopolska province in 2005 (tonnes per year)

## Socio-economic health factors in Wielkopolska region

Table 4 shows demographic indices for the region of Wielkopolska as recorded in the period of 2005–2009. Population density did not change over the period under study, totalling 114 people per sq kilometre as from 2007 and 2009. Urban communes have population density of over 1,500 persons/km, while rural communes

Table 4. Basic demographic indices by commune type Wielkopolska province, 2005–2009

| Specification | Type of commune | 2005 | 2006 | 2007 | 2008 | 2009 |
|---|---|---|---|---|---|---|
| Population per 1 km$^2$ of total area | total | 113 | 113 | 114 | 114 | 114 |
| | urban | 1551 | 1563 | 1557 | 1551 | 1547 |
| | urban-rural | 88 | 88 | 88 | 89 | 89 |
| | rural | 58 | 59 | 59 | 60 | 60 |
| | urban areas | 1300 | 1292 | 1289 | 1286 | 1285 |
| | rural areas | 51 | 51 | 52 | 52 | 53 |
| Feminisation ratio | total | 106 | 106 | 106 | 106 | 106 |
| | urban | 112 | 112 | 112 | 112 | 112 |
| | urban-rural | 104 | 104 | 104 | 104 | 104 |
| | rural | 101 | 101 | 101 | 101 | 101 |
| | urban areas | 110 | 111 | 111 | 111 | 111 |
| | rural areas | 101 | 101 | 101 | 101 | 101 |

Table 4. continue

| Specification | Type of commune | 2005 | 2006 | 2007 | 2008 | 2009 |
|---|---|---|---|---|---|---|
| Post-productive population (in % of total population). No data for 2008 and 2009 | total | 13.8 | 14.2 | 14.4 | | |
| | urban | 15.4 | 16.0 | 16.5 | | |
| | urban-rural | 12.9 | 13.2 | 13.4 | | |
| | rural | 12.8 | 13.0 | 13.1 | | |
| | urban areas | 14.5 | 15.0 | 15.5 | | |
| | rural areas | 12.9 | 13.1 | 13.1 | | |
| Live births per 1,000 population | total | 10.5 | 10.8 | 11.3 | 12.1 | 12.0 |
| | urban | 9.6 | 9.9 | 10.2 | 10.8 | 11.1 |
| | urban-rural | 10.9 | 11.1 | 11.9 | 12.7 | 12.6 |
| | rural | 11.2 | 11.6 | 11.8 | 12.9 | 12.4 |
| | urban areas | 9.9 | 10.1 | 10.6 | 11.3 | 11.7 |
| | rural areas | 11.2 | 11.7 | 12.1 | 13.1 | 12.5 |
| Deaths per 1,000 population | total | 9.0 | 9.1 | 9.2 | 9.2 | 9.3 |
| | urban | 9.2 | 9.4 | 9.5 | 9.5 | 9.7 |
| | urban-rural | 8.9 | 9.0 | 9.0 | 9.0 | 9.2 |
| | rural | 8.8 | 9.0 | 9.0 | 8.9 | 8.9 |
| | urban areas | 9.0 | 9.2 | 9.3 | 9.3 | 9.5 |
| | rural areas | 9.0 | 9.0 | 9.0 | 9.0 | 9.1 |
| Infant deaths per 1,000 population. No data for 2008 and 2009 | total | 0.06 | 0.07 | 0.08 | | |
| | urban | 0.05 | 0.07 | 0.07 | | |
| | urban-rural | 0.08 | 0.07 | 0.08 | | |
| | rural | 0.06 | 0.07 | 0.09 | | |
| | urban areas | 0.06 | 0.07 | 0.07 | | |
| | rural areas | 0.06 | 0.07 | 0.09 | | |
| Population growth rate per 1,000 population. No data for 2008 and 2009 | total | 1.5 | 1.7 | 2.1 | | |
| | urban | 0.4 | 0.5 | 0.7 | | |
| | urban-rural | 2.0 | 2.1 | 2.9 | | |
| | rural | 2.4 | 2.6 | 2.8 | | |
| | urban areas | 0.9 | 0.8 | 1.3 | | |
| | rural areas | 2.2 | 2.7 | 3.1 | | |
| Internal and international net migration for permanent residence per 1,000 population. No data for 2008 and 2009 | total | 0.68 | 0.16 | 0.55 | | |
| | urban | –2.70 | –3.42 | –4.31 | | |
| | urban-rural | 0.81 | 0.66 | 1.56 | | |
| | rural | 5.49 | 4.62 | 6.04 | | |
| | urban areas | –1.97 | –2.68 | –3.19 | | |
| | rural areas | 4.18 | 3.84 | 5.37 | | |

are below the level of 60 persons/km. The highest population density levels were observed in the communes of central Wielkopolska, particularly those bordering on Poznań and other major cities of the region (Fig. 7).

Gender distribution, not unlike population density, remained unchanged throughout the studied period. From 2005, the ratio was 106 females per 100 males. However, gender proportions vary according to a commune type. In urban communes, the feminisation ratio is 112, while rural communes are characterised with more balanced gender distribution, with 101 females per 100 males. As shown in Figure 8, the highest values of feminisation ratio were found in big cities and their surrounding communes, with over 106 females per 100 males. On the other hand, rural communes in the northern part of Wielkopolska province showed the feminisation ratio of less than 100.

In terms of age distribution, a tendency was observed for the post-productive population to grow systematically, although not very rapidly. In 2005, this age group accounted for 13.8% of the total province population, while in 2007 for as much as 14.4%. In relation to geographic distribution and types of administrative units, the highest proportion of post-productive population is found in the south-west of Wielkopolska and in the cities, where it reaches even 16% (Fig. 9). The rate is markedly lower (10%) in three communes near Poznań – Suchy Las, Czerwonak and Murowana Goślina – reflecting effects of internal migrations. This relates to the progressing suburbanisation process and the migration of young people from Poznań to the adjacent communes due to lower real estate prices.

An equally systematic and small growth in live birth rate was observed. A total number of live births per 1,000 population increased in 2005–2009 from 10.5 to 12.0 (by 1.5). More children are born in villages and rural areas as compared to cities and urban areas. The geographical distribution of birth rates indicates a division of the region into the northern part, with 12 live births per 1,000 population, and the southern part characterised by a lower birth rate (below 10) (Fig. 10).

The death rate in Wielkopolska province in the period 2005–2009 remained at a similar level (Table 4). The overall ratio was 9 deaths per 1,000 population but it was higher in the cities (9.7 per 1,000 population in 2009). The highest death rate was recorded in the communes of the south-eastern Wielkopolska (Fig. 11). Similar death rate levels of over 11 deaths per 1,000 population occur also in many of the region's cities. Notably, however some of the communes neighbouring those cities were marked with very low death rates. This is clearly exemplified by the capital city of Wielkopolska province, which is surrounded by a ring of communes with some of the lowest death rates in the region.

The values of infant death rate increased in the studied period from 0.06 in 2005 to 0.08 in 2007. In urban areas the rate was lower than in rural areas. The geographical distribution of this indicator showed that communes with highest infant mortality per 1,000 population were situated in the northern and eastern part of Wielkopolska, whereas towns and communes around the city of Poznań were characterised with distinctly lower infant death rates (Fig. 12). This is thought to be associated with better health care, both in terms of medical equipment and access to medical services.

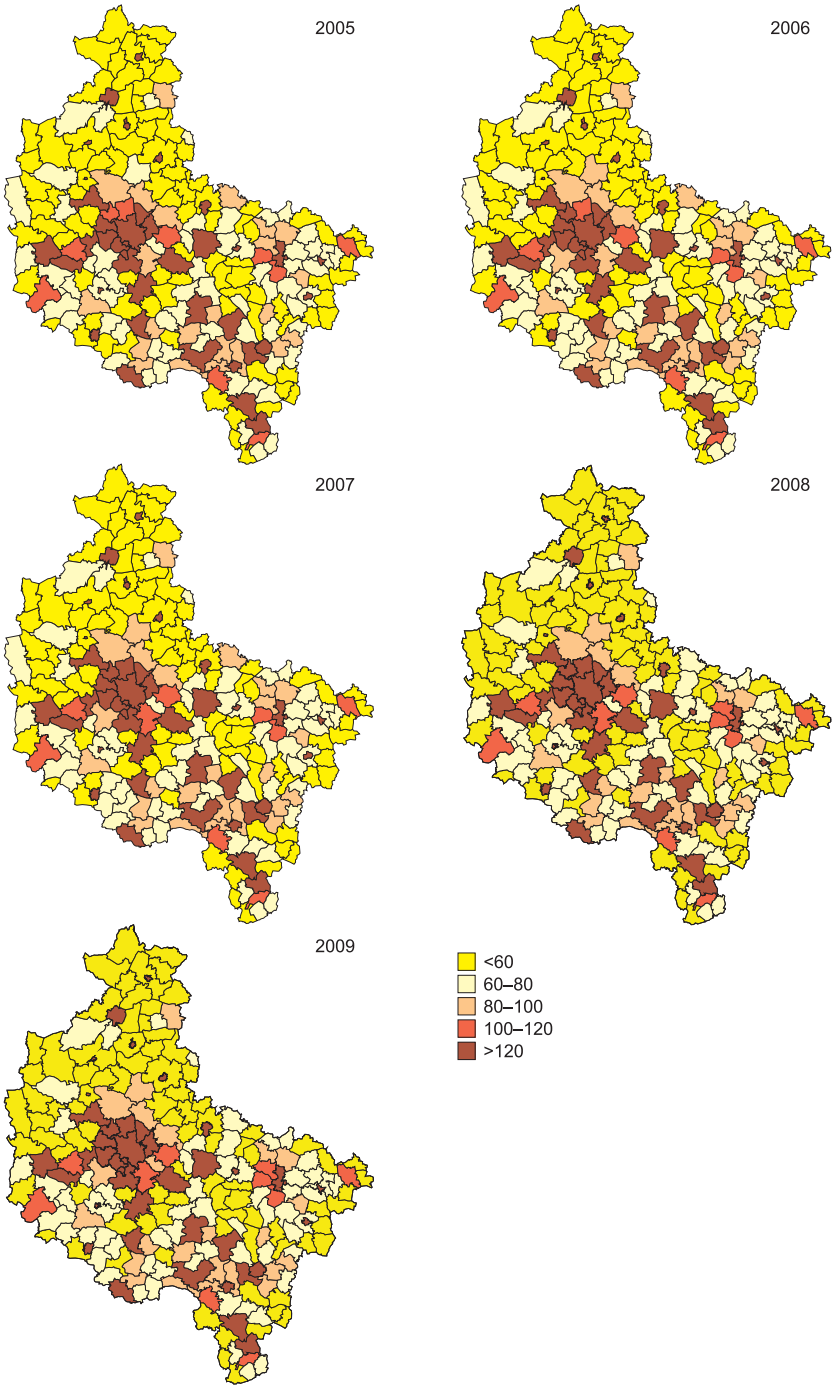



Fig. 7. Population density (population per 1 km$^2$ of total area) in communes of Wielkopolska region. 2005–2009

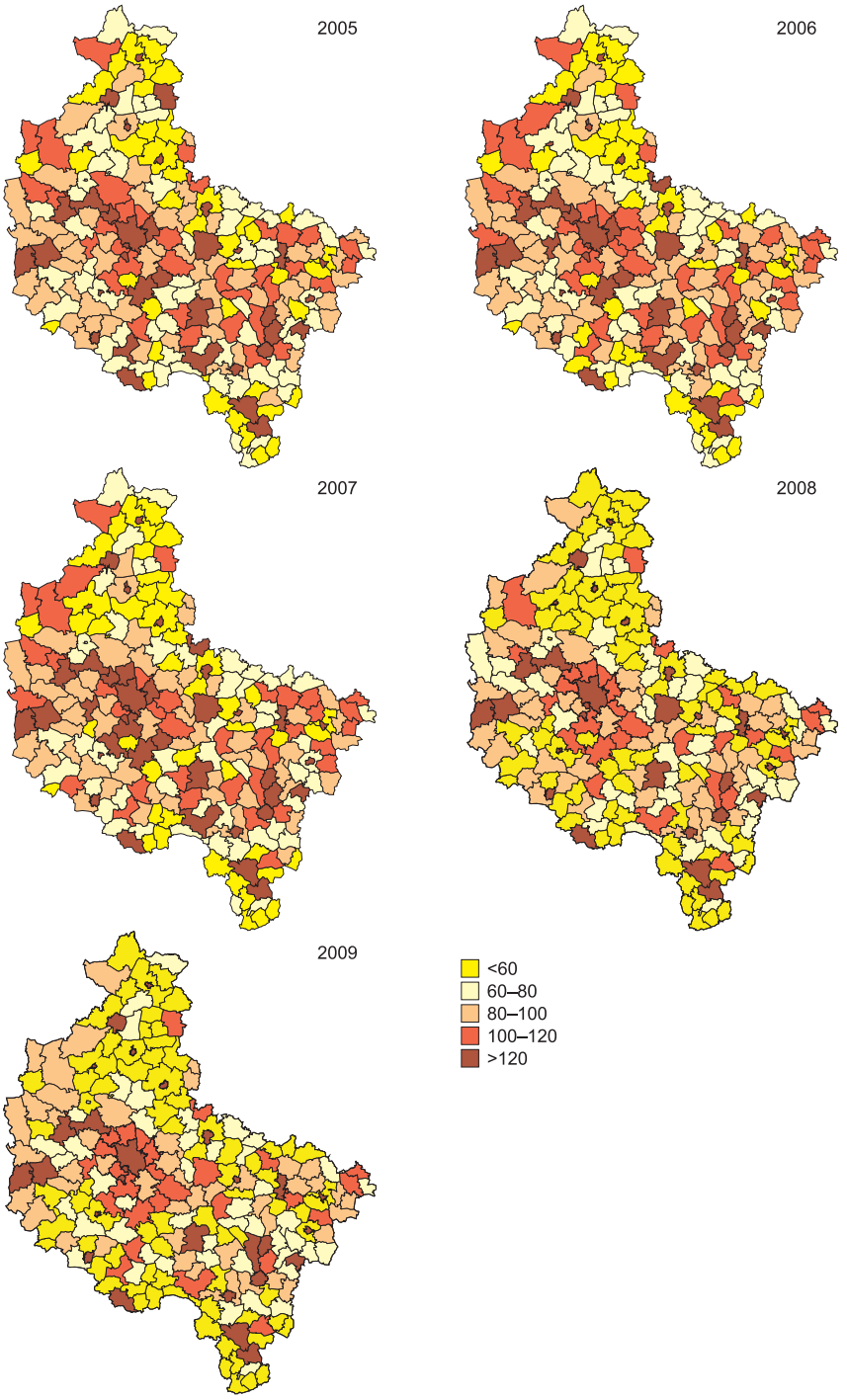



Fig. 8. Feminisation rate in communes of Wielkopolska province, 2005–2009

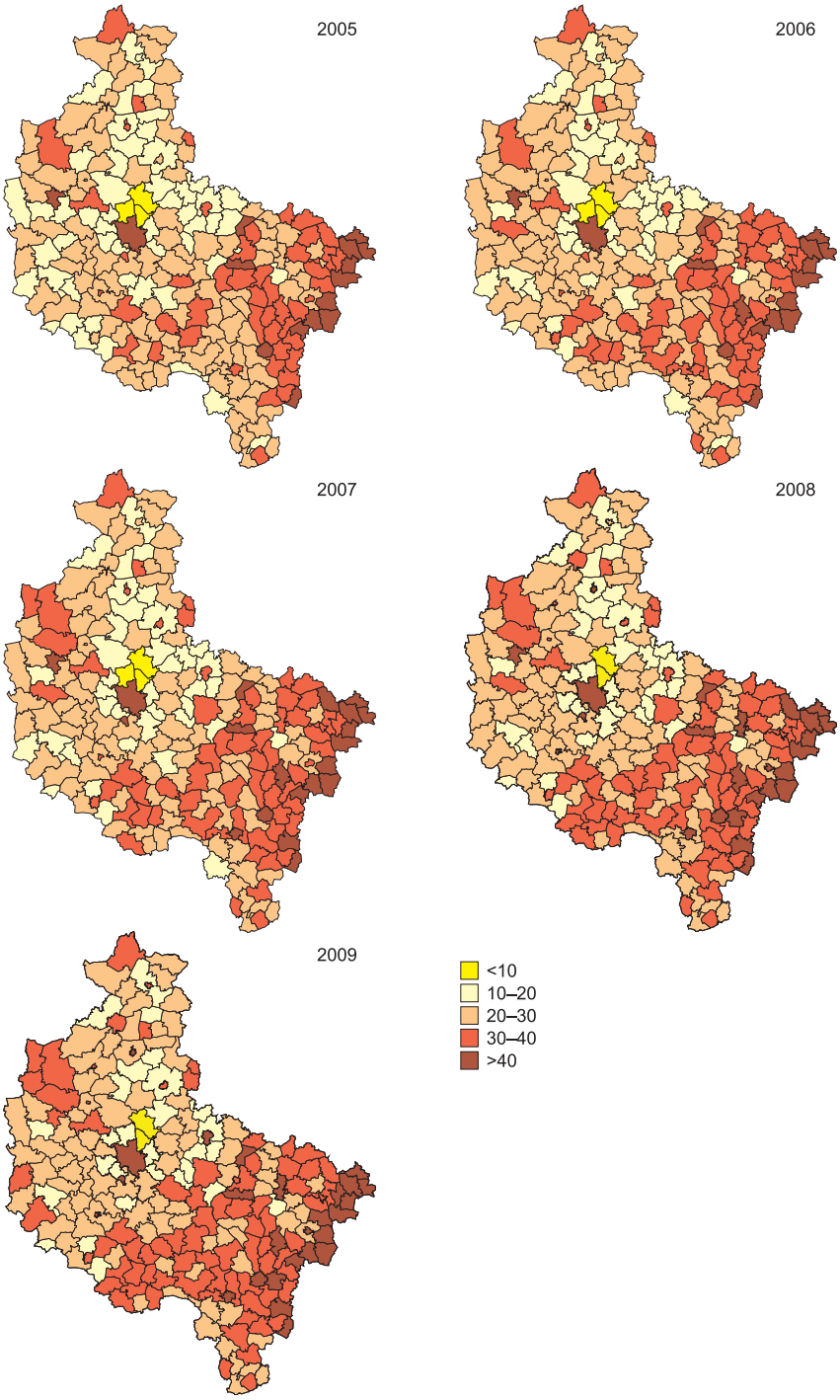



Fig. 9. Percentage of post-productive population in Wielkopolska province, 2005–2009

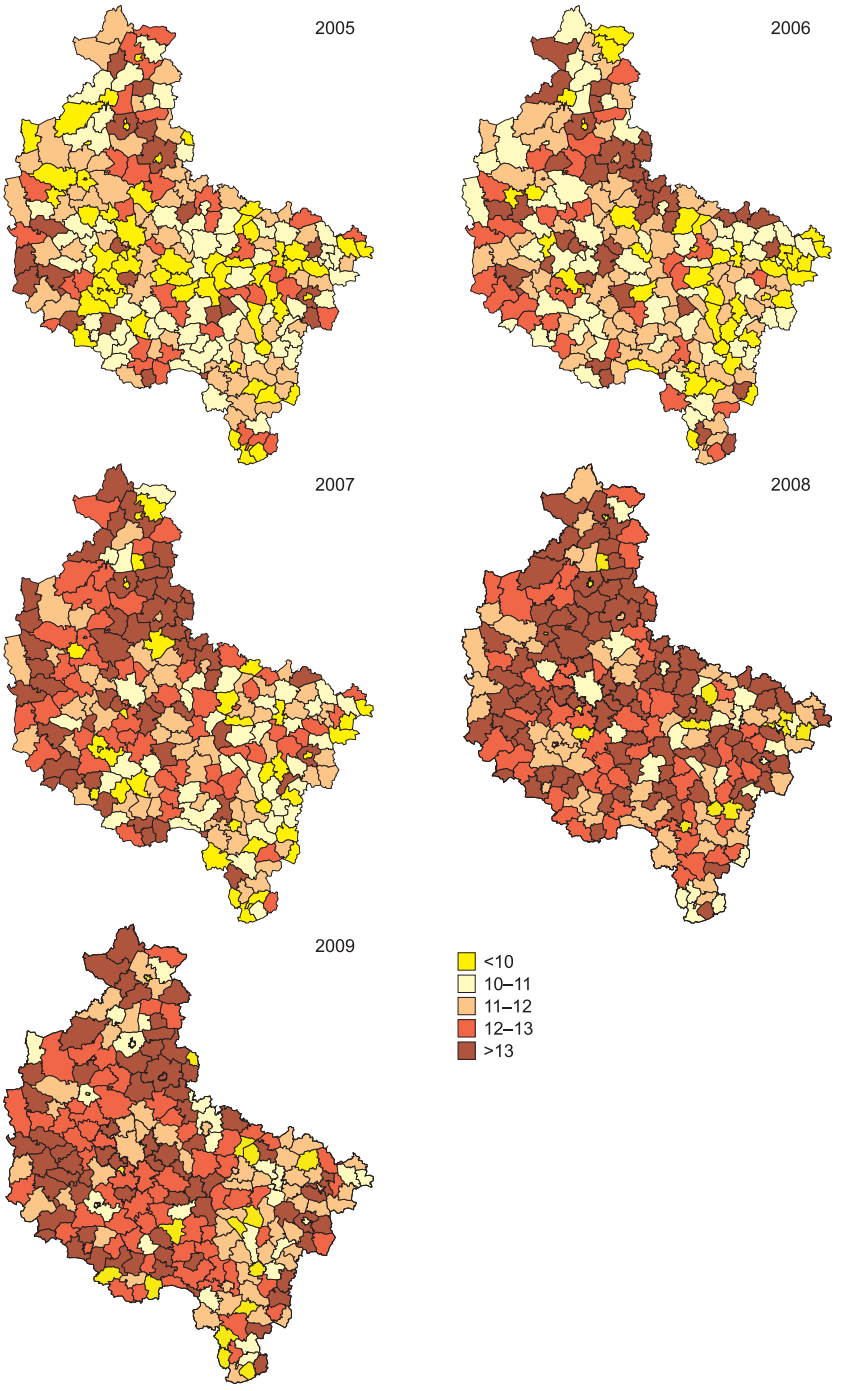



Fig. 10. Live births in communes of Wielkopolska province, 2005–2009 (per 1,000 population)

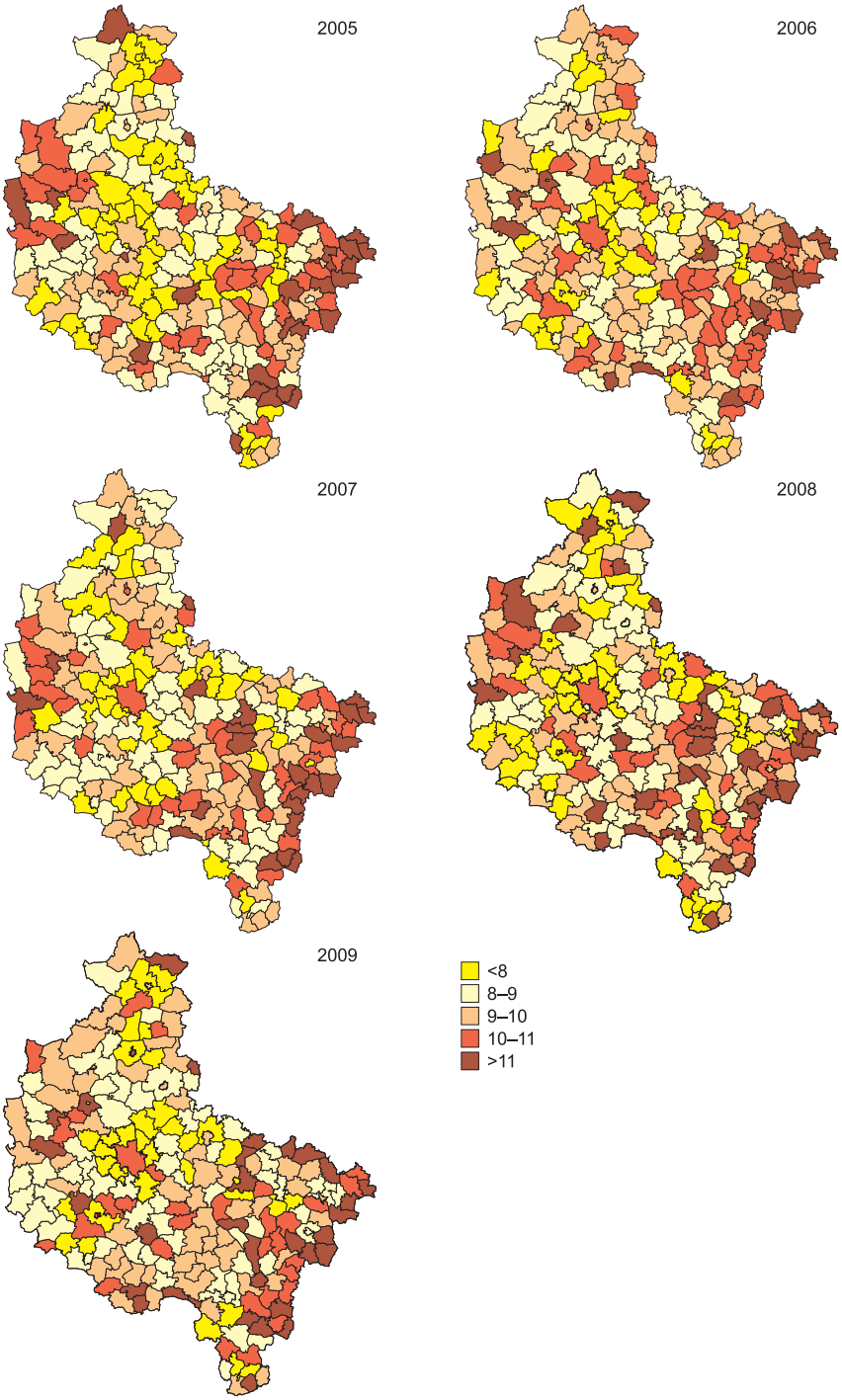



Fig. 11. Death rate in Wielkopolska province, 2005–2009 (per 1,000 population)

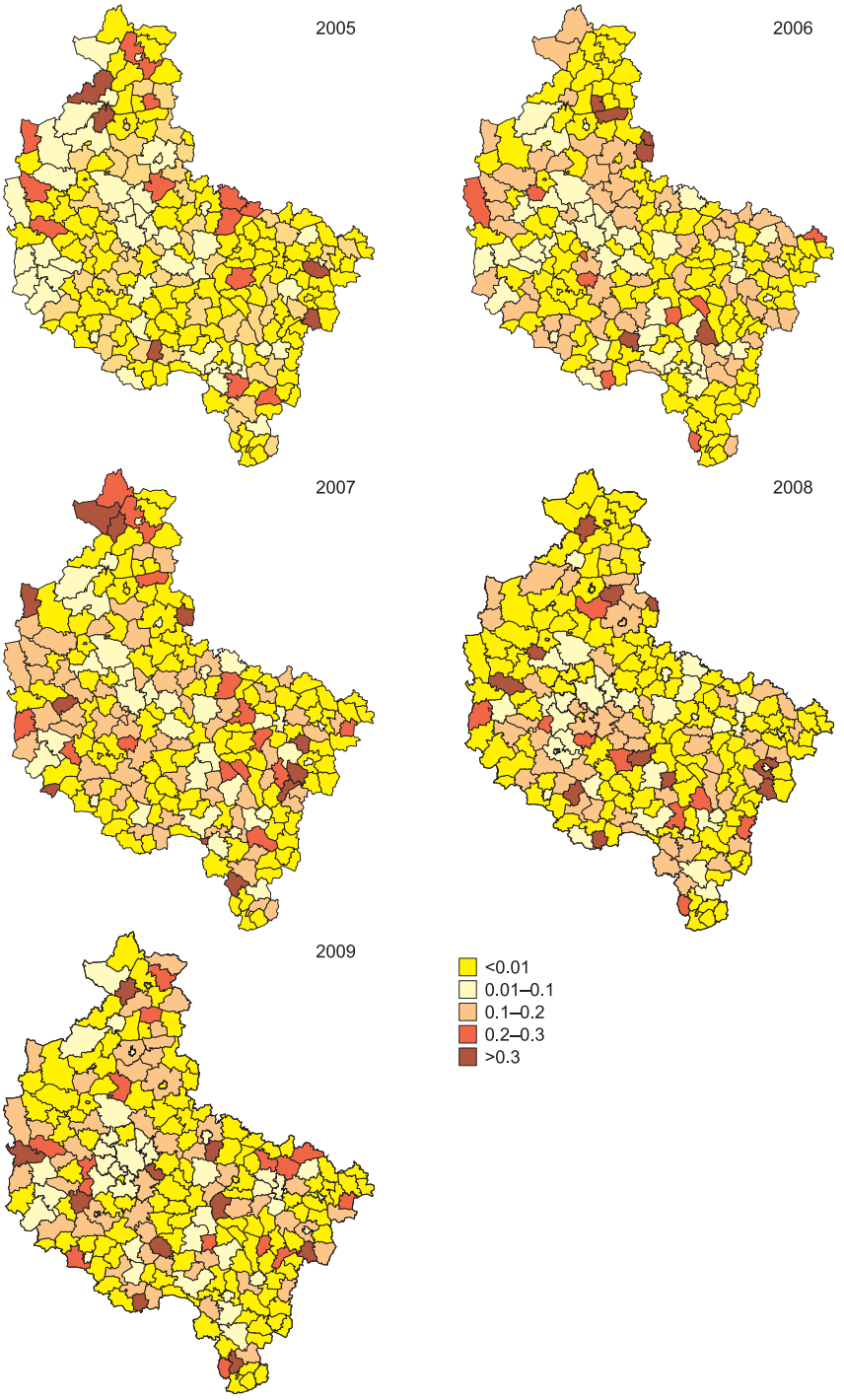



Fig. 12. Infant death rate in Wielkopolska province, 2005–2009 (per 1,000 population)

The period of 2005–2009 saw a significant increase in the population growth rate (difference between the overall number of live births and the number of deaths) per 1,000 population (Table 4 and Fig. 13). High growth rate was observed primarily in urban-rural communes. mainly their rural parts and rural communes, where it nearly reached the level of 3. In urban communes, the level was below 1. More children were born in the country than in the city and, even though infant mortality in urban areas is lower. It is rural areas that show higher population growth. The geographical distribution of population growth rates showed higher figures in the communes of northern Wielkopolska (nearly 4 persons per 1,000 population), while the southern part of the province recorded a negative growth rate. Notably, while bigger cities had negative population growth rates, their neighbouring communes were observed to grow at the rate of up to 6 persons per 1,000 population.

Interesting conclusion may be derived from the analysis of the process of internal migration. Overall, in 2005–2007, net migration in the province of Wielkopolska was positive (Table 4). This was mostly accounted for by a high positive net migration in rural communes (nearly 5 persons per 1,000 population). Urban communes recorded a negative migration balance due to the outflow of urban population to the country (over 4 persons per 1,000 population in 2007). The process of suburbanisation is particularly pronounced in district capitals (Fig. 14). Poznań, Konin and Leszno showed negative migration rates, while positive migration was recorded in communes surrounding those cities.

Due to the absence of data on mean life expectancy in relation to communes,the analysis was limited to subregions and the years 2007 and 2009 (Table 5). In 2007, mean life expectancy in Wielkopolska province was 79 years for females and 70 years for males. Two years later, females' life expectancy increased by almost a year, while males' life expectancy by 2 years (in 2009, mean life expectancy for females was 79.9 years and for males 72 years). The regional distribution broken down into subregions does not show significant differences. The longest lifespans for females were observed in the urban subregions of Kalisz, Konin and Poznań, and the city of Poznań itself. The longest lifespans for males were recorded in the city of Poznań and the urban subregions of Leszno and Poznań.

Selected living condition indices were compiled in Table 6. Average usable floor space of dwellings per person in Wielkopolska province grew slightly in the studied period by 1.2 $m^2$, from 23.7 $m^2$ in 2005 to 24.9 $m^2$ in 2009 (Table 6). A similar growth was observed in the cities (23.4 $m^2$ in 2005 to 25.2 $m^2$ in 2009) and in the village (from 24.1 $m^2$ to 25.4 $m^2$). The rate did not differ significantly across commune types oscillating around the average. Relatively better housing standards (larger floor area per person) were found in the communes of Poznań district and in the southern part of Wielkopolska (Fig. 15). In those communes, average usable floor space per person exceeded 27 $m^2$. Whereas, communes located in the north of the region were characterised by smaller average usable floor space of 21 $m^2$.

A rapid economic growth in Poland and Wielkopolska, as well as the upturn in the mortgage market and housing industry brought the rise in the number of newly built apartments. In 2005, there were 3 apartments built per 1,000 population to

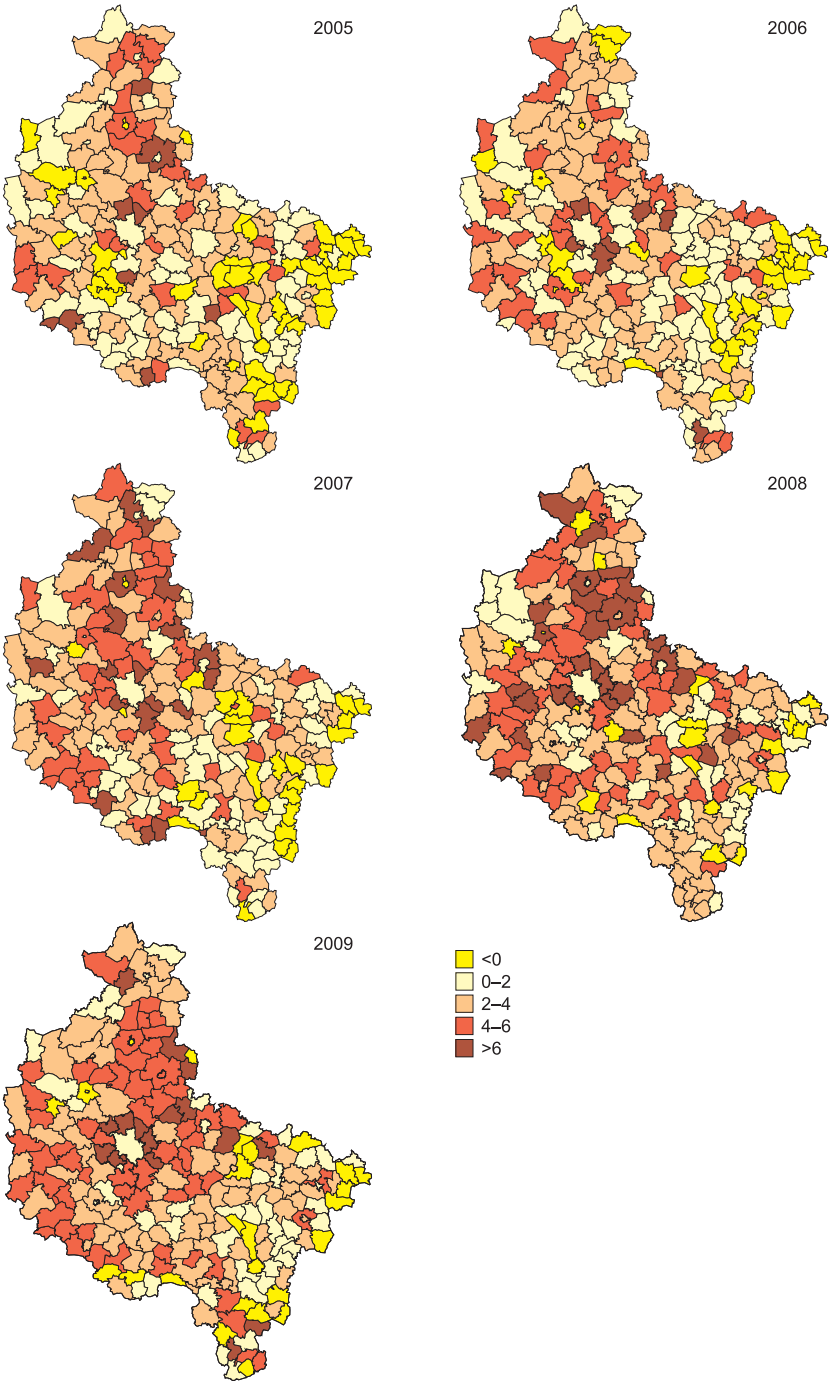



Fig. 13. Population growth rate in Wielkopolska province, 2005–2009 (per 1,000 population)

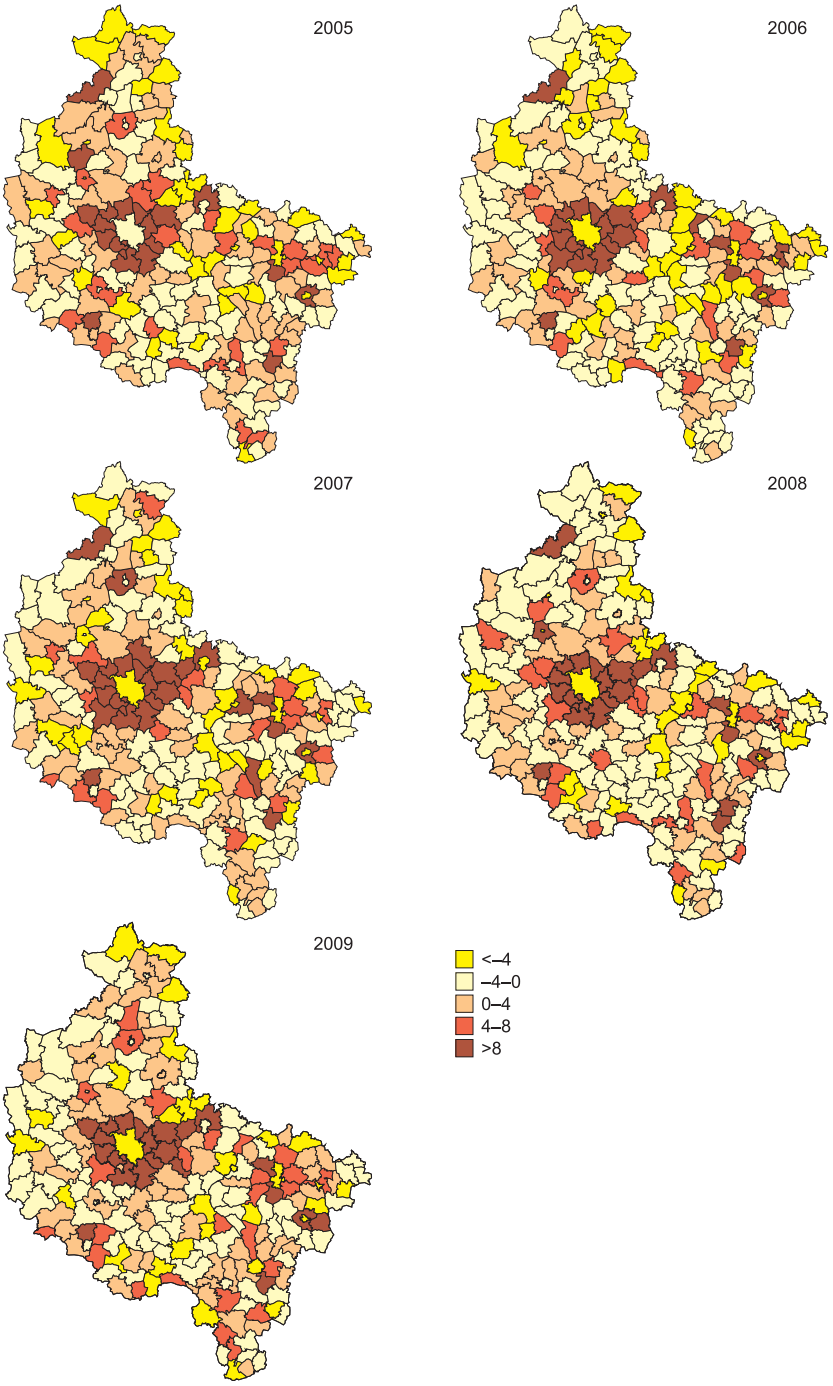



Fig. 14. Net migration in communes of Wielkopolska province by commune types. 2005–2009 (per 1,000 population)

Table 5. Mean life expectancy (in years) at birth by Wielkopolska subregions

| Subregion | 2007 | | 2009 | |
|---|---|---|---|---|
| | Males | Females | Males | Females |
| Kalisz | 70.9 | 79.6 | 71.3 | 80.2 |
| urban area | 71.6 | 79.4 | 71.5 | 80.3 |
| rural area | 70.3 | 79.9 | 71.2 | 80.1 |
| Konin | 70.8 | 79.7 | 71.5 | 80.2 |
| urban area | 71.6 | 79.7 | 72.6 | 80.1 |
| rural area | 70.1 | 79.8 | 70.5 | 80.3 |
| Leszno | 71.6 | 79.0 | 72.0 | 78.8 |
| urban area | 72.1 | 79.1 | 72.8 | 79.1 |
| rural area | 71.2 | 78.8 | 71.3 | 78.6 |
| Piła | 70.1 | 79.3 | 71.1 | 79.1 |
| urban area | 70.9 | 79.6 | 71.0 | 79.2 |
| rural area | 69.2 | 79.0 | 71.2 | 79.2 |
| Poznań | 71.7 | 79.1 | 72.4 | 79.6 |
| urban area | 72.1 | 78.5 | 72.5 | 79.8 |
| rural area | 71.4 | 79.8 | 72.4 | 79.4 |
| Poznań | 73.3 | 79.6 | 73.5 | 80.9 |

Table 6. Selected indices of Wielkopolska population's living conditions in 2005–2009 by commune types

| Specification | Type of commune | 2005 | 2006 | 2007 | 2008 | 2009 |
|---|---|---|---|---|---|---|
| Dwelling stocks | total | 23.7 | 23.8 | 24.0 | 24.6 | 24.9 |
| Average usable floor space of dwellings per person in $m^2$ | urban | 23.4 | 23.6 | 23.9 | 24.9 | 25.2 |
| | urban-rural | 23.3 | 23.4 | 23.6 | 23.9 | 24.1 |
| | rural | 24.1 | 24.2 | 24.4 | 25.2 | 25.4 |
| | urban areas | 24.2 | 24.3 | 24.5 | 24.7 | 24.8 |
| | rural areas | 23.6 | 23.7 | 23.8 | 24.5 | 24.6 |
| Dwellings built per 1,000 population. No data for 2008 and 2009 | total | 3.5 | 3.1 | 4.2 | | |
| | urban | 4.4 | 3.7 | 5.1 | | |
| | urban-rural | 2.7 | 2.4 | 3.2 | | |
| | rural | 3.6 | 3.5 | 4.5 | | |
| | urban areas | 3.9 | 3.2 | 4.4 | | |
| | rural areas | 3.1 | 3.1 | 4.0 | | |
| Population using water-supply system (in % of total population) | total | 91.7 | 91.9 | 92.1 | 92.3 | 92.4 |
| | urban | 96.4 | 96.8 | 96.8 | 96.9 | 97.0 |
| | urban-rural | 89.8 | 90.1 | 90.1 | 90.5 | 90.6 |
| | rural | 87.6 | 88.0 | 88.1 | 88.5 | 88.8 |
| | urban areas | 96.1 | 96.4 | 96.4 | 96.6 | 96.6 |
| | rural areas | 85.8 | 86.3 | 86.3 | 86.8 | 87.0 |
| Population using sewerage system (in % of total population) | total | 58.0 | 58.6 | 59.2 | 59.7 | 60.4 |
| | urban | 88.3 | 88.3 | 88.5 | 88.9 | 89.2 |
| | urban-rural | 48.1 | 49.3 | 50.3 | 50.8 | 51.6 |
| | rural | 27.7 | 28.9 | 30.0 | 31.3 | 32.8 |
| | urban areas | 84.6 | 84.9 | 85.2 | 85.7 | 86.1 |
| | rural areas | 22.6 | 24.0 | 25.3 | 26.2 | 27.6 |

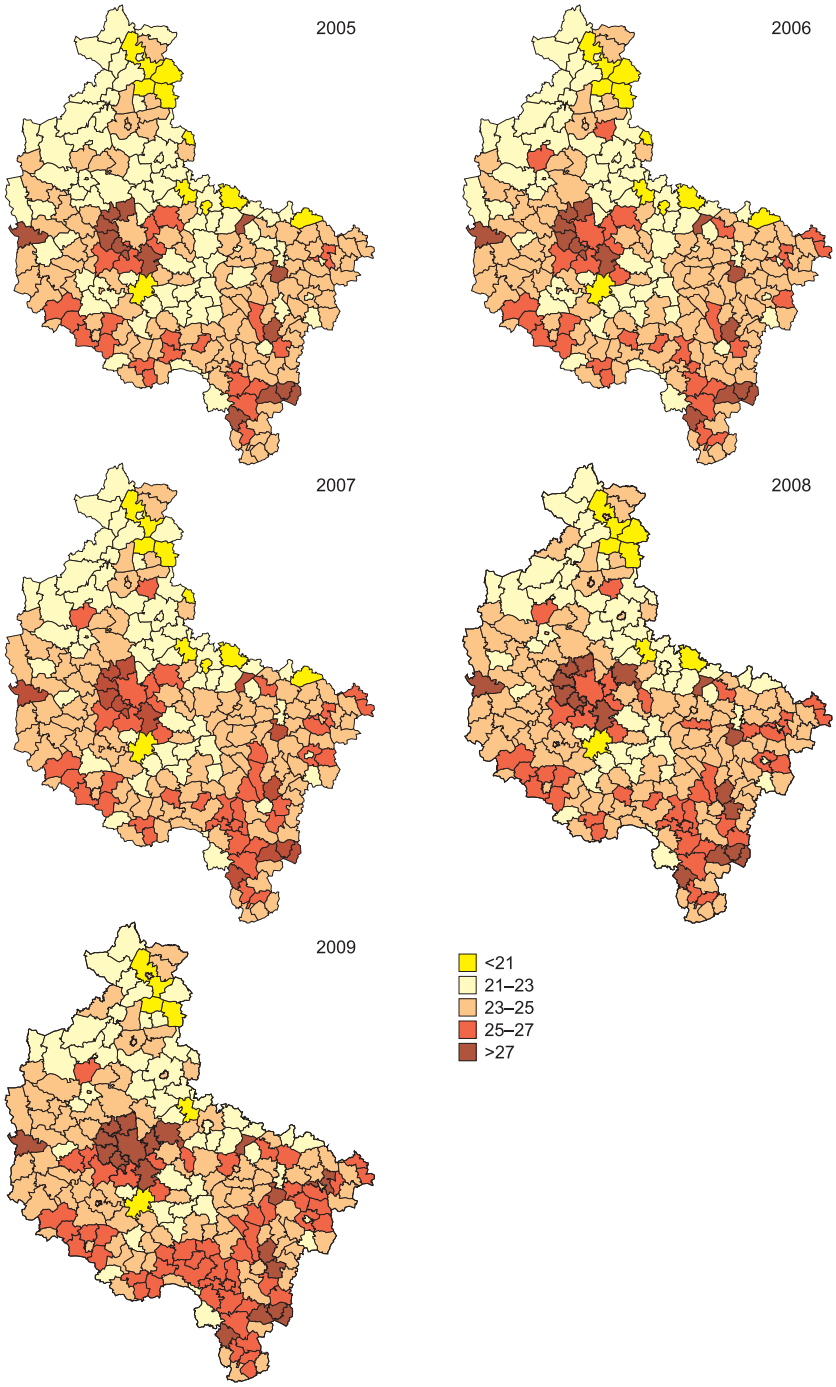



Fig. 15. Average usable floor space of dwellings per person in communes of Wielkopolska province, 2005–2009 (in sq. metres per person)

go up to 4 in 2007 (Table 6). The highest number of dwellings was constructed in urban areas (5.1 per 1,000 population in 2007), and the lowest number in urban-rural communes (3.2 per 1,000 population in 2007). In the analysed period, the housing market was particularly strong in communes of central Wielkopolska, mostly in the district of Poznań (Fig. 16). Each year, more than 4 apartments per 1,000 population were built there. Remarkably, the towns of Komorniki and Swarzędz recorded as many as 24 new dwellings per 1,000 population. Housing development was much more limited in the northern part of the province with meaningful examples of the communes of Łobżenica and Wysoka where no apartments were build during 2007.

The proportion of population using the water supply network in Wielkopolska province was high, amounting to nearly 92% throughout the studied period (Table 6). The rate was found to be almost 97% in the cities and over 90% in rural communes. Insufficient water supply systems can be found in the peripheries of the province (Fig. 17). The proportion of population with access to water supply network in those regions was below 80%.

The proportion of population using sewerage systems remains significantly lower than for water supply. Less than 60% of Wielkopolska population have access to the sewage disposal services (Table 6). Even in the cities the rate is only 89%. The situation of rural areas improved, however, with the proportion of population with access to sewage infrastructure rising from 28% in 2005 to 33% in 2009. The best conditions in this respect were found in the communes of central and northern Wielkopolska (Fig. 18), with 70% of population utilising sewage infrastructure. In the south-east of the region the rate was below 20%.

Table 7 shows population income levels. Communes' income from taxes rose from PLN 428 per person in 2005 to PLN 610 per person in 2007. Their levels highly diversified across commune types (Table 7). In urban communes, income

Table 7. Wielkopolska population's income levels in 2005–2009 by commune types

| Specification | Type of commune | 2005 | 2006 | 2007 | 2008 | 2009 |
|---|---|---|---|---|---|---|
| Communal income from tax (PLN per capita). No data for 2008 and 2009 | total | 428.8 | 494.2 | 610.2 | | |
| | urban | 725.6 | 830.9 | 1014.8 | | |
| | urban-rural | 272.0 | 319.2 | 400.1 | | |
| | rural | 234.6 | 273.0 | 350.3 | | |
| Percentage of registered unemployed persons to total productive population | total | 9.7 | 7.7 | 5.1 | 4.1 | 6.0 |
| | urban | 8.0 | 6.4 | 4.2 | 3.3 | 5.1 |
| | urban-rural | 10.6 | 8.4 | 5.5 | 4.6 | 6.7 |
| | rural | 11.0 | 8.7 | 6.0 | 4.6 | 6.4 |
| Expenditure on housing benefits (PLN per capita). No data for 2008 and 2009. | total | 31.3 | 31.5 | 27.9 | | |
| | urban | 47.5 | 48.9 | 43.6 | | |
| | urban-rural | 28.3 | 28.4 | 25.3 | | |
| | rural | 11.8 | 11.0 | 9.1 | | |

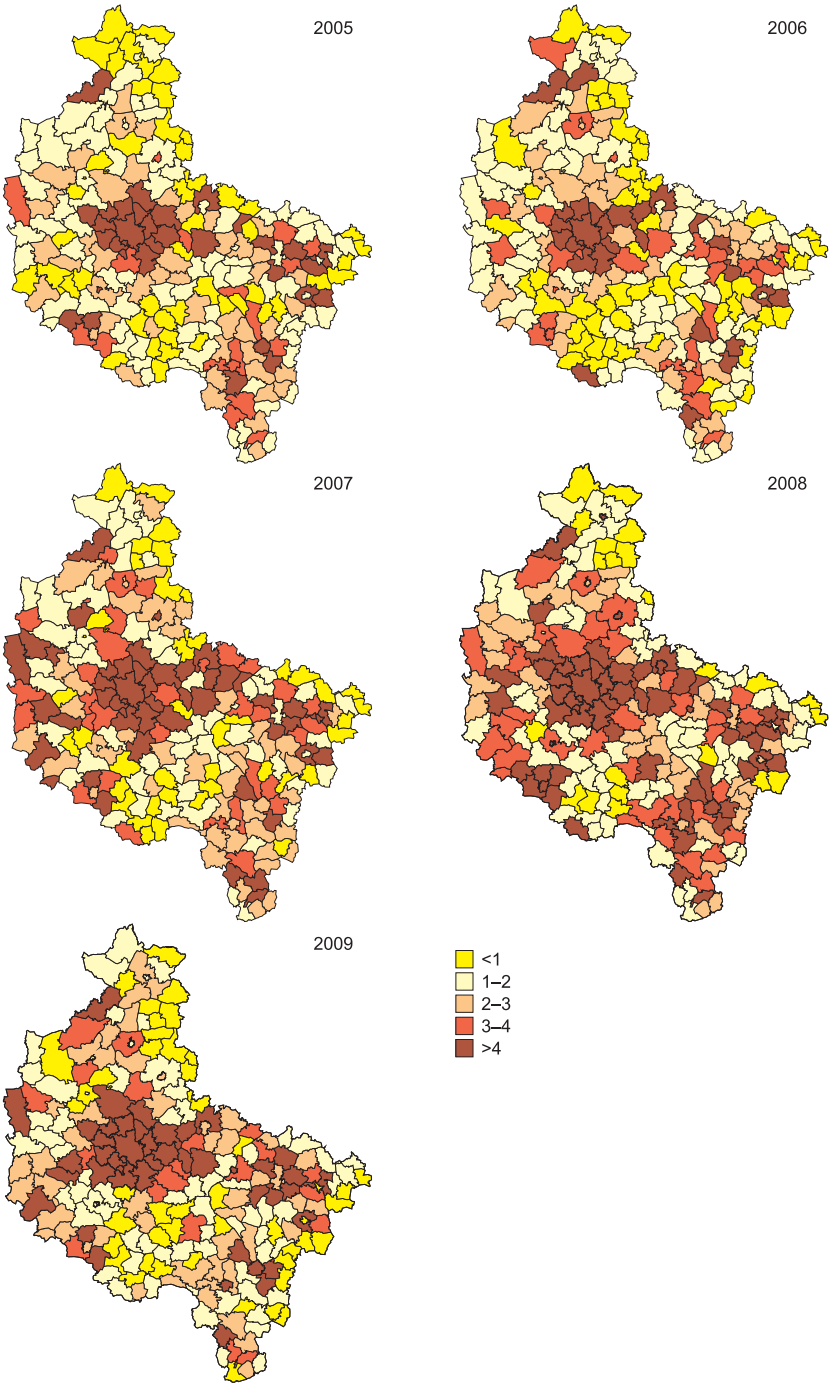



Fig. 16. Number of newly built dwellings in communes of Wielkopolska province, 2005–2009 (per 1,000 population)

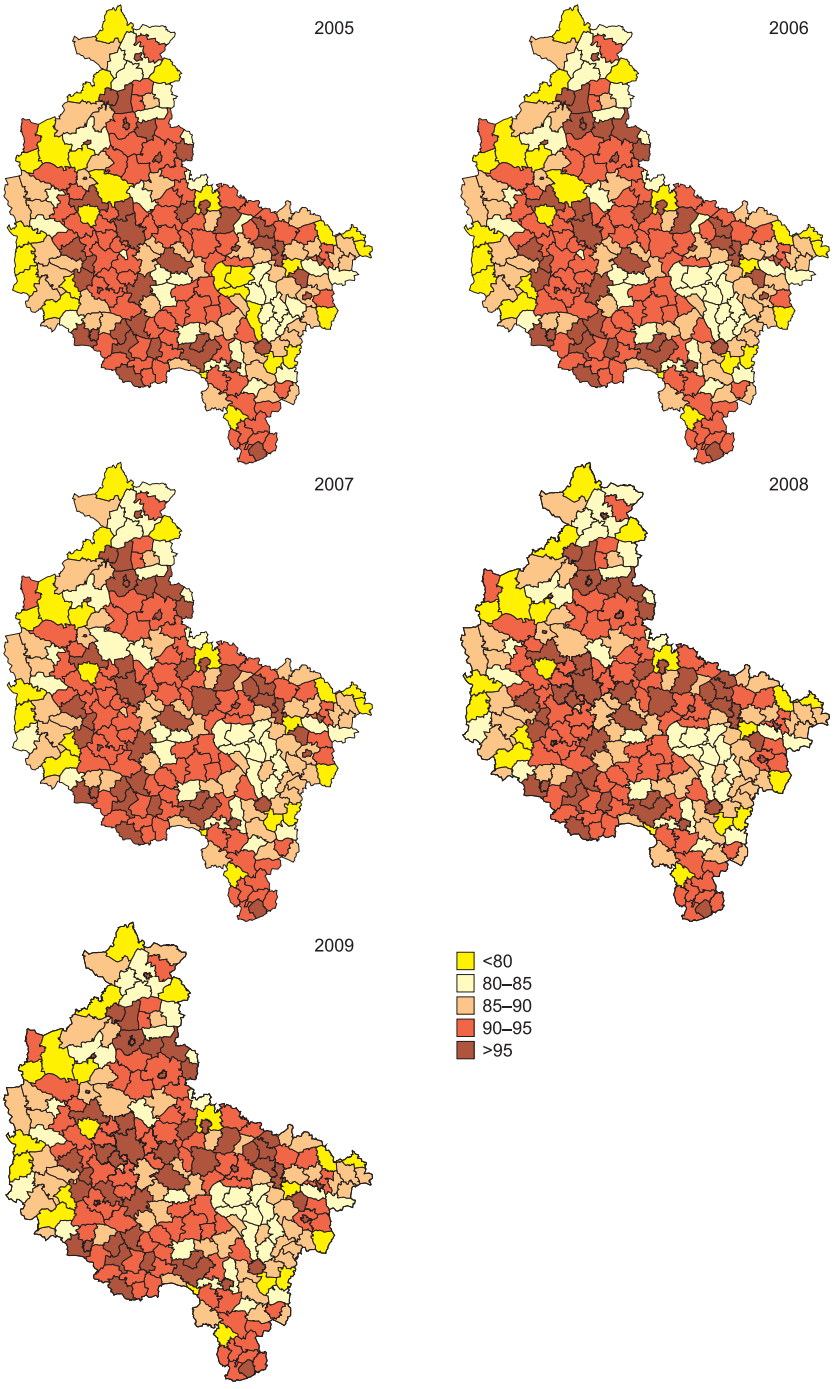



Fig. 17. The proportion of population using water supply network in communes of Wielkopolska province, 2005–2009 (percent of total population)

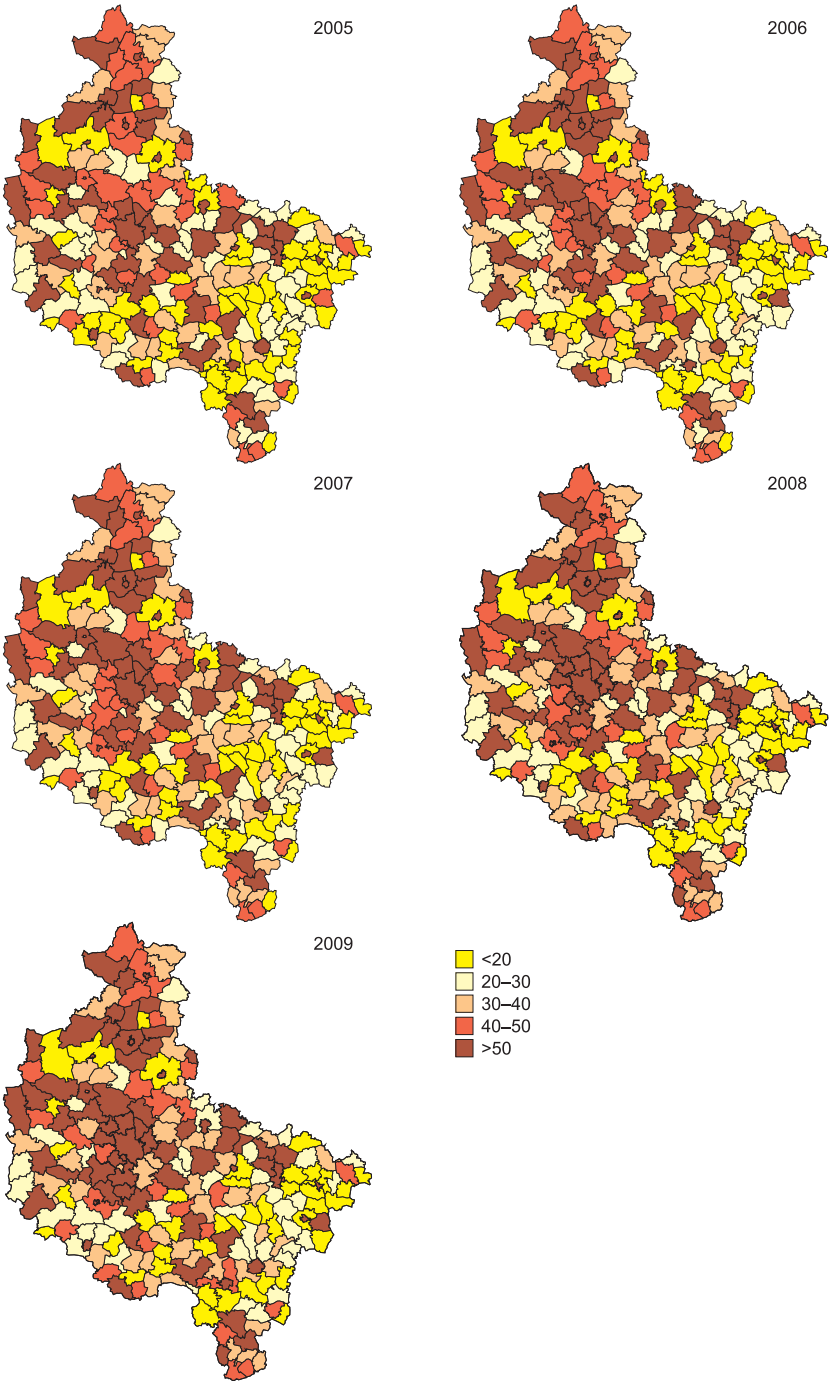



Fig. 18. The proportion of population using the sewage system in communes of Wielkopolska province, 2005–2009 (percent of total population)

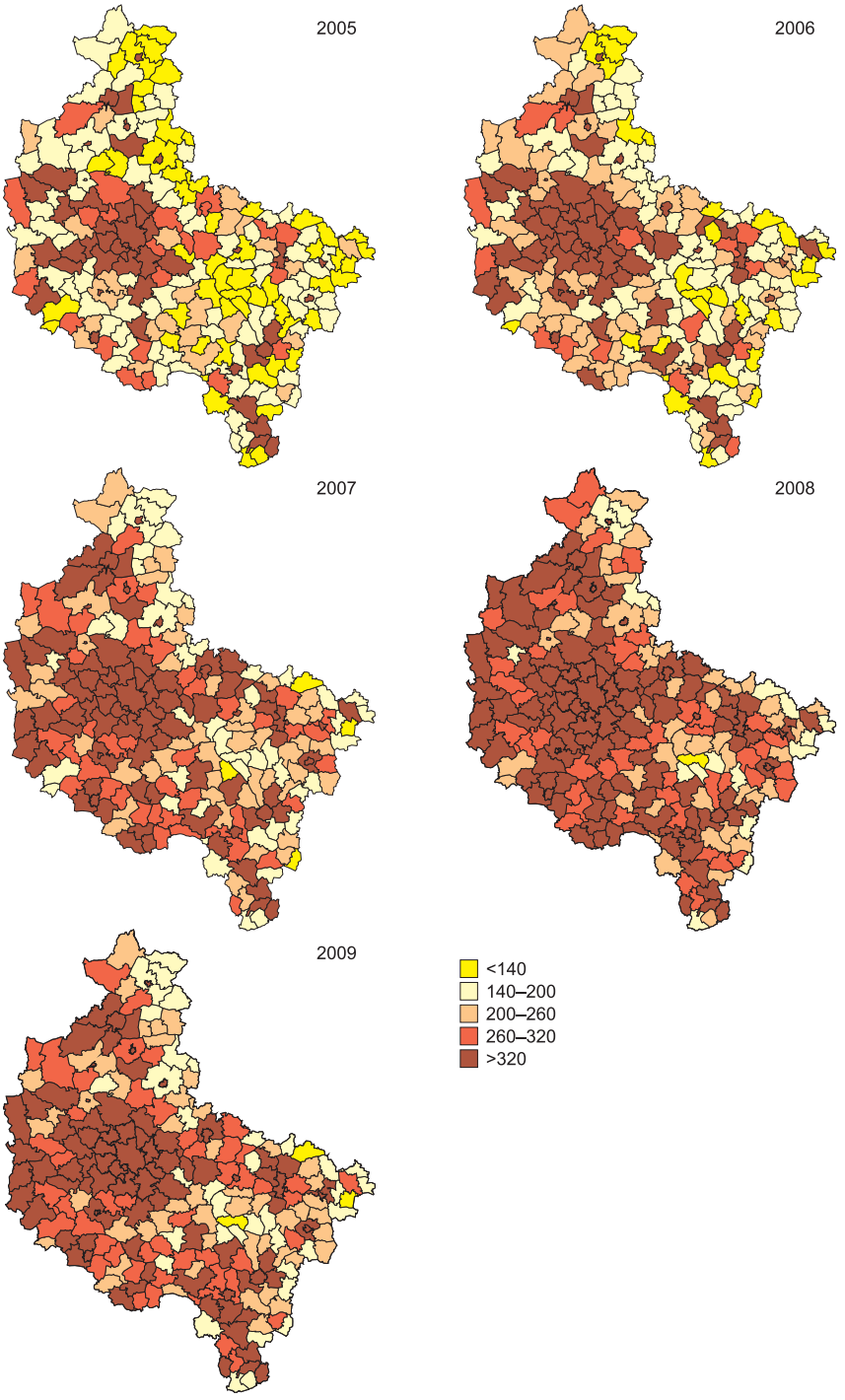



Fig. 19. Communes' income from taxes (in PLN per capita)

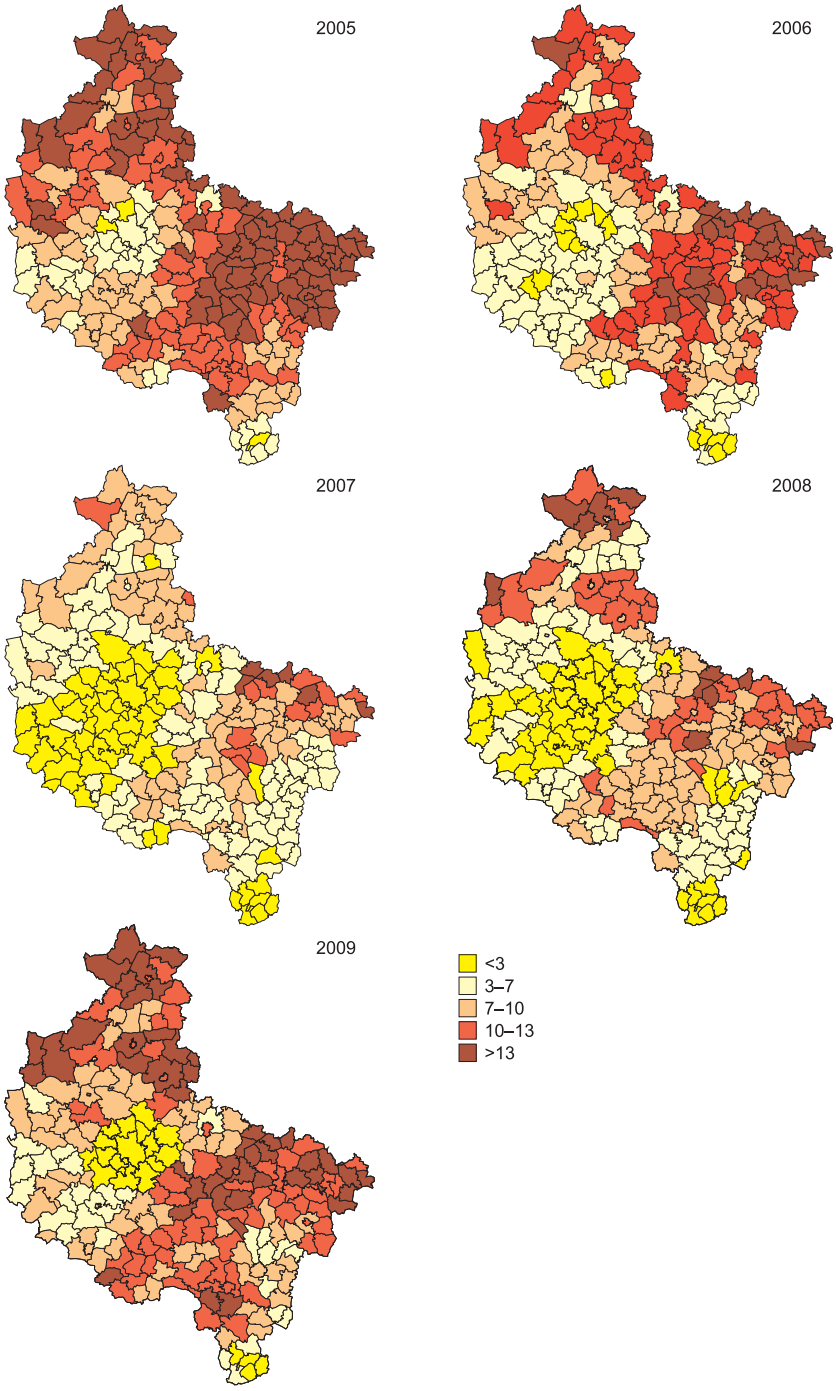



Fig. 20. The proportion of unemployed in relation to productive population (percent of total population)

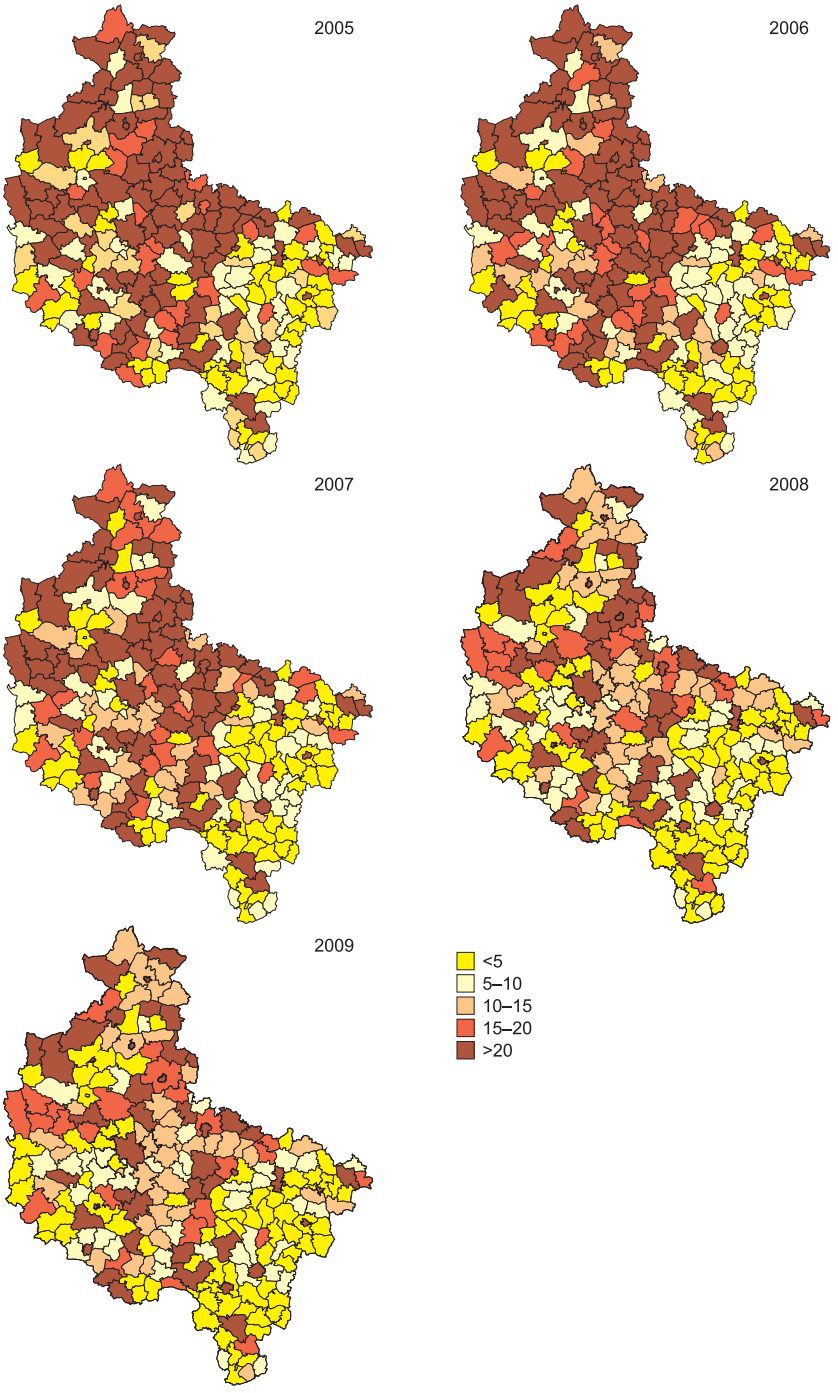



Fig. 21. Housing benefit expenditure in communes of Wielkopolska province, 2005–2009 (PLN per capita)

from that source in 2007 amounted to PLN 1,000 per capita and was much higher than in urban-rural communes (PLN 400) and rural communes (PLN 350). Apart from urban communes, high income levels from personal and corporate income taxes were recorded in all types of communes located in Poznań district and in the western part of the province. In the communes of south-eastern Wielkopolska, income levels were up to ten times lower, estimated at below PLN 140 per capita. Distribution on communes' income from taxes is shown in Figure 19.

Intensive economic growth in Poland was associated not only with growth of communes' income but also with decrease of unemployed people. In Wielkopolska province, in 2005 the number of unemployed people consisted of 10% of productive age population, and in 2007 only 5%. This tendency was observed in all communes. In 2007, the highest unemployment was observed in rural communes. Across all studied period, the lowest unemployment rate, approx. 4% was observed in communes located in the central part of Wielkopolska province and increased with the distance from Poznań. The distribution of unemployment rat is show in Figure 20.

Housing benefits are an example of social support provided by communes to their lowest income residents. The amount of benefits paid in Wielkopolska province fell from PLN 31 per capita to PLN 27.9 per capita (Table 7). This form of support was used in larger extent by urban population. with PLN 43 paid per person in 2007. High income from taxes combined with high level of housing benefits may indicate a large spread of income levels in urban population. In rural communes, the average amount of housing benefit was below PLN 10 per person. The geographical distribution of this form of financial support reveals higher levels in communes located in the central part (except the district of Poznań) and in the northern part of the province (over PLN 20 per capita), and lower levels in the eastern part of Wielkopolska (less than PLN 5 zł per capita) (Fig. 21).

## The classification of Wielkopolska communes in terms of natural and socio-economic health factors – multidimensional perspective

The analysis comprised 19 above described indices characterising natural and socio-economic health factors in communes of Wielkopolska province in 2007 and 2009. Except for the pairs of variables $x_{11}$:$x_8$ (r=0.78) and $x_{11}$:$x_9$ (r=–0.7), the study did not find any high or significant correlations. Five clusters comprising various numbers of communes were distinguished using the *k-mean* method. The consecutive results of the classification procedure for 2009 are shown in Tables 8 and 9. The distribution of communes are shown on map in Figure 22. Five clusters indicated health-related environmental conditions ranging from the worst (marked as I) to the best (marked as V) conditions.

The first cluster includes the city of Poznań only. The natural environmental conditions of Poznań are detrimental to human health. This results above all from

Table 8. Indicators of natural and socio-economic health factors in communes of Wielkopolska province in 2009 taken to multidimensional analysis

| Variable | Indicator | Mean | SD |
|---|---|---|---|
| $x_1$ | Proportion of forests (in % of commune land area) | 21.9 | 14.8 |
| $x_2$ | Proportion of legally protected areas (in % of commune land area) | 29.1 | 32.2 |
| $x_3$ | Waste collected (in tonnes per year) | 3580.3 | 14,566.2 |
| $x_4$ | Population density (per 1 km) | 191.5 | 423.4 |
| $x_5$ | Feminisation Ratio | 102.3 | 3.7 |
| $x_6$ | Live births (per 1,000 population) | 12.4 | 1.8 |
| $x_7$ | Deaths (per 1,000 population) | 9.4 | 1.8 |
| $x_8$ | Population growth (per 1,000 population) | 3.0 | 2.6 |
| $x_9$ | Proportion of post-productive population (in % of total population) | 13.9 | 1.8 |
| $x_{10}$ | Net migration (per 1,000 population) | 1.6 | 8.5 |
| $x_{11}$ | Average usable floor space of dwelling (in $m^2$ per capita). | 24.6 | 2.5 |
| $x_{12}$ | New apartments (per 1,000 population) | 3.1 | 3.9 |
| $x_{13}$ | Proportion of population with access to water supply system (in % of population) | 89.5 | 6.8 |
| $x_{14}$ | Proportion of population with access to sewage system (in % of population) | 40.1 | 23.4 |
| $x_{15}$ | Communes tax income (PLN per capita). | 366.7 | 231.7 |
| $x_{16}$ | Proportion of unemployed persons to total productive population (in %) | 6.9 | 2.6 |
| $x_{17}$ | Expenditure on housing benefits (in PLN per capita) | 12.9 | 13.5 |
| $x_{18}$ | Infant deaths (per 1,000 population) | 0.1 | 0.1 |
| $x_{19}$ | Green areas (in % of commune area) | 0.2 | 0.4 |

Source: Based on own study.

a low proportion of forest and protected areas and huge amounts of waste produced. In terms of socio-economic conditions, the evaluation is more ambiguous. On one hand, some adverse phenomena are observed, with Poznań having the largest post-productive population, high death rate and low birth rate, although population growth rate is still positive. On the other hand however, residents of Poznań enjoy relatively good housing conditions. Average floor area is larger than anywhere else in the province as is the number of newly built apartments. More than 90% of the city population have access to sewage disposal services. Poznań has a very high level of tax income and low unemployment rate indicating its population to be wealthier than the rest of the province. On the other hand, high amounts spent on housing benefits indicate a large income disparity.

The second cluster includes 8 communes and five cities (*inter alia* Gniezno, Kalisz, Konin, Ostrów Wielkopolski and Piła) and Poznań's suburban urban-rural commune of Swarzędz. These localities are on one hand characterised by small proportions of forests and green areas, high rate of post-production population, low population growth, small-sized apartments and high unemployment but on

Table 9. Descriptive statistics of five obtained clusters

| Variable | Cluster I | | Cluster II | | Cluster III | | Cluster IV | | Cluster V | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| $x_1$ | 13.8 | 0.0 | 13.3 | 15.0 | 15.1 | 11.4 | 23.9 | 17.0 | 22.6 | 14.4 |
| $x_2$ | 1.4 | 0.0 | 6.9 | 14.2 | 23.6 | 30.6 | 29.4 | 27.2 | 30.8 | 33.8 |
| $x_3$ | 209626.6 | 0.0 | 23784.6 | 5877.7 | 8552.9 | 1833.0 | 3496.1 | 1148.8 | 806.5 | 541.2 |
| $x_4$ | 2117.0 | 0.0 | 1164.6 | 676.6 | 727.2 | 889.1 | 231.9 | 394.6 | 66.8 | 62.4 |
| $x_5$ | 115.0 | 0.0 | 109.5 | 2.6 | 107.3 | 3.0 | 103.9 | 2.7 | 101.0 | 2.9 |
| $x_6$ | 11.3 | 0.0 | 11.5 | 1.4 | 12.4 | 1.1 | 12.4 | 1.3 | 12.4 | 2.0 |
| $x_7$ | 10.1 | 0.0 | 8.9 | 1.4 | 8.7 | 1.1 | 8.8 | 1.6 | 9.7 | 1.9 |
| $x_8$ | 1.2 | 0.0 | 2.6 | 2.5 | 3.8 | 2.1 | 3.6 | 2.2 | 2.7 | 2.8 |
| $x_9$ | 18.7 | 0.0 | 15.8 | 2.0 | 14.2 | 1.5 | 13.4 | 1.6 | 13.9 | 1.8 |
| $x_{10}$ | –5.3 | 0.0 | –1.2 | 4.8 | 6.1 | 16.2 | 4.0 | 12.8 | 0.7 | 5.4 |
| $x_{11}$ | 27.2 | 0.0 | 24.1 | 2.1 | 25.3 | 4.1 | 24.8 | 3.3 | 24.4 | 2.0 |
| $x_{12}$ | 6.5 | 0.0 | 4.4 | 3.5 | 6.2 | 8.7 | 4.7 | 5.4 | 2.2 | 1.9 |
| $x_{13}$ | 96.4 | 0.0 | 97.0 | 1.5 | 94.9 | 3.5 | 91.8 | 4.7 | 87.9 | 7.0 |
| $x_{14}$ | 90.2 | 0.0 | 84.8 | 11.6 | 73.3 | 16.2 | 55.4 | 18.4 | 30.3 | 16.6 |
| $x_{15}$ | 1509.2 | 0.0 | 733.2 | 190.2 | 664.2 | 303.5 | 486.9 | 237.1 | 280.7 | 128.4 |
| $x_{16}$ | 2.8 | 0.0 | 6.6 | 2.1 | 6.0 | 2.8 | 6.3 | 3.1 | 7.2 | 2.4 |
| $x_{17}$ | 21.6 | 0.0 | 40.3 | 16.8 | 22.8 | 15.3 | 19.8 | 16.3 | 8.9 | 8.9 |
| $x_{18}$ | 0.0 | 0.0 | 0.1 | 0.0 | 0.1 | 0.0 | 0.1 | 0.1 | 0.1 | 0.1 |
| $x_{19}$ | 1.7 | 0.0 | 0.9 | 0.6 | 0.7 | 0.9 | 0.2 | 0.4 | 0.1 | 0.1 |

Source: Based on own study.

the other hand they have a very high percentage of households using water supply and sewage systems and high tax income levels.

The third cluster includes 16 communes – urban communes (*inter alia* Leszno and Turek), urban-rural communes (Krotoszyn, Szamotuły, Śrem, Środa Wielkopolska, Wolsztyn and Września) and rural commune (Tarnowo Podgórne). Small proportions of forest and green areas need to be pointed out for these communes as main unfavourable health factors, while housing standards and well-developed infrastructure are their strong assets.

The fourth cluster comprises 41 communes located mainly in the central and western part of Wielkopolska. These include predominantly urban-rural communes but also urban and rural. Those units are marked with larger areas of forests, very low proportion of post-productive population, high population growth but also a relatively high unemployment rate and low levels of tax income.

The last cluster encompasses 160 communes (mostly rural). They have particularly favourable natural conditions, with large woodlands and protected areas. The population growth rate is high with large number of births but also deaths. However, residents of those communes suffer from lower housing standards and limited access to water supply and sewage disposal infrastructure. These communes are also characterised by high unemployment.

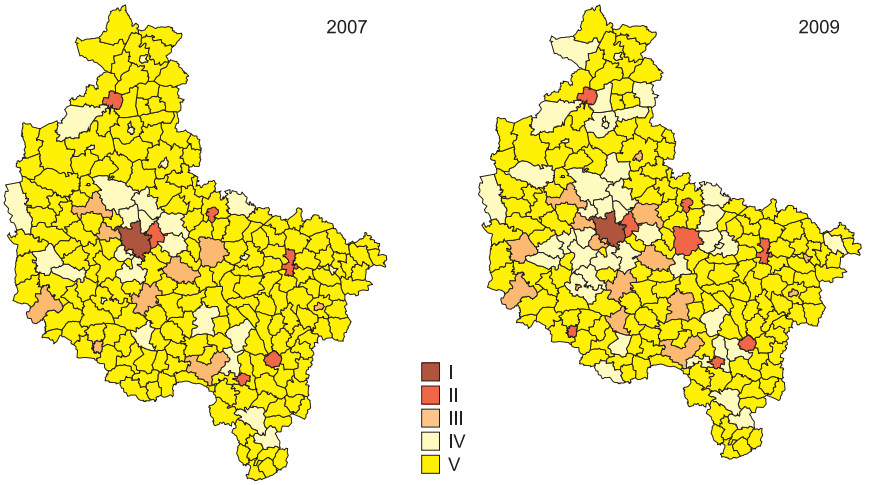



Fig. 22. Classification of Wielkopolska communes in terms of natural and socio-economic health factors in 2007 and 2009. Results of multivariable approach. Five clusters I – the worst

## Concluding statements

Human health is determined by a variety of concomitant factors originating from natural built and socio-economic environments. A detailed analysis of those factors and their mutual interactions allows to identify areas with high risk of morbidity.

The highly urbanised and industrialised regions of Wielkopolska province are most disadvantaged in terms of natural health factors. The bioclimate of those areas is detrimental to health mostly due to adverse effects of human activity. As regards socio-economic conditions, it has to be noted that communities of the cities and some urban-rural communes, including Krotoszyn, Śrem, Wolsztyn or Swarzędz and Tarnowo Podgórne (rural commune) enjoy better housing standards and higher income levels. Urban communes, however, are more affected by the process of population ageing.

The communes with lower urbanisation and industrialisation levels, mostly rural, feature lower extent of natural environment degradation with bioclimate neutral to human organism. They also have and additional benefit of large forest and legally protected areas. On the other hand, those communities struggle with poor housing standards and low income levels which have possible negative impact on health. These tendencies are not confined to the region of Wielkopolska.

Cities around Poznań show an excess of females over males, while in rural areas the proportion tends to be reversed. This demographic indicator demonstrates both the continuous extension of life expectancy and the ageing of Wielkopolska population. These trends are commonly observed in most EU countries.

The world is growing more and more urbanised, with the proportion of urban population steadily increasing. Wielkopolska region, on the other hand, shows the process of suburbanisation that is particularly clear around Poznań. The population migrating from the city is likely to improve their natural health conditions.

## Acknowledgments

Authors are grateful to Paweł Motek, Faculty of Geographical and Geological Sciences, Adam Mickiewicz University, who developed figures based on Regional Data Bank of the Central Statistical Office (GUS).

## References

*Achieving health equity: from root causes to fair outcomes*. WHO 2007:17–40.
Available at www.who.int/socialdeterminants/resources/csdh_media/csdh_interim_statement_07.pdf. Accessed: February 24, 2011.
Anderson W.T.: Lifestyle and psychographics: a critical review and recommendation. *Adv Consum Res* 1984;11:405–411.
Bielicki T., Welon Z.: Growth data as indicators of social inequalities: the case of Poland. *Yearbook Phys Anthropol* 1982;25:153–167.
Bielicki T., Waliszko H.: Urbanization-dependent gradients in stature among Polish conscripts in 1976 and 1986. *Am J Hum Biol* 1991; 3:419–424.
Centers for Disease Control and Prevention. The Burden of Chronic Diseases and Their Risk Factors: National and State Perspectives 2004. Atlanta: U.S. Department of Health and Human Services; 2004. Available at: http://www.cdc.gov/nccdphp/burdenbook2004 Accessed: February 24, 2011.
Chojnicki Z., Czyż T.: *Metody taksonomii numerycznej w regionalizacji geograficznej*. 1973. Warszawa: PWN.
Chojnicki Z., Czyż T.: Charakterystyka małych miast regionu poznańskiego a koncepcja kontinuum miejsko-wiejskiego. In: P. Korceli and A. Gawryszewski (Ed.), *Współczesne przemiany regionalnych systemów osadniczych w Polsce*. 1989; 152:139–157. Prace Geograficzne, IGiPZ PAN.
Danker-Hopfe H.: Menarcheal age in Europe. *Am J Phys Anthropol* 1986; 29(S7):81–112.
Edgar A., Sedgwick P.: *Key Concepts in Cultural Theory*. 1999. London and New York: Routledge.
Farat R.: (Ed.) *Atlas klimatu województwa wielkopolskiego*. 2004. Instytut Meteorologii i Gospodarki Wodnej Oddział w Poznaniu, Poznań.
Holzer J.: *Demografia*. 1999. Warszawa: Polskie Wydawnictwo Ekonomiczne.
Kondracki J.: *Geografia regionalna Polski*. 1998. Warszawa: Wydawnictwo Naukowe PWN.
Kozłowska-Szczęsna T, Krawczyk B.: Klimatyczne uwarunkowania zdrowotności w Polsce. In: L. Mazurkiewicz and A. Wróbel (Ed.) *Przestrzenne problemy zdrowotności*, 1990. Conference Papers 9, Warszawa: 72–86.
Kozłowska-Szczęsna T., Błażejczyk K., Krawczyk B.:, *Bioklimat człowieka. Metody i ich zastosowanie w badaniach bioklimatu Polski*. 1997. Warszawa: Instytut Geografii i Przestrzennego Zagospodarowania PAN.

Kozłowska-Szczęsna T., Krawczyk B., Kuchcik M.: *Wpływ środowiska atmosferycznego na zdrowie i samopoczucie człowieka.* 2004. Warszawa: Instytut Geografii i Przestrzennego Zagospodarowania im. Stanisława Leszczyckiego PAN.

Lalonde M.: A new perspective on the health of Canadians a working document. Cat. No. H31-1374 ISBN 0-662-50019-9. Ottawa, April 1974.

Marsili D.: Environmental health and the multidimensional concept of development: the role of environmental epidemiology within international cooperation initiatives research from animal testing to clinical experience. *Ann Ist Super Sanità* 2009; 45(1):76–82.

Malinowska M.: Bioklimaty województwa pomorskiego. In: T. Michalski (Ed.), *Zróżnicowanie przestrzenne sytuacji zdrowotnej, systemu bezpieczeństwa i usług medycznych w województwie pomorskim.* 2002; 5:38–44. Regiony Nadmorskie, Wydawnictwo EJB.

Mazurkiewicz L., Wróbel A.: *Przestrzenne problemy zdrowotności*. 1999. Conference Papers 9, Warszawa.

Michalski T.: Uwarunkowania sytuacji zdrowotnej ludności wiejskiej w Polsce. In: J. Bański and E. Rydz (Ed.) *Społeczne problemy wsi.* 2002; 2:31–39. Warszawa: Studia obszarów wiejskich.

Michalski T.: Zdrowie środowiskowe w województwie pomorskim-ujęcie przestrzenne. In: T. Michalski (Ed.), *Zróżnicowanie przestrzenne sytuacji zdrowotnej, systemu bezpieczeństwa i usług medycznych w województwie pomorskim*. 2002a; 5:45–49. Regiony Nadmorskie, Wydawnictwo EJB.

Mitchell R., Popham F.: Effect of exposure to natural environment on health inequalities: an observational population study. *Lancet* 2008; 372(9650):1655–1660.

Motek P.: *Gospodarka finansowa samorządu terytorialnego w województwie wielkopolskim.* 2006. Poznań: Bogucki Wydawnictwo Naukowe;

Parsons K.C.: *Human thermal environments: the effects of hot, moderate, and cold*
*environments on human health, comfort and performance*. 2003. London, New York: Taylor&Francis.

Parysek J.: 1982. *Modele klasyfikacji w geografii*. 1982. Poznań: Uniwersytet im. Adama Mickiewicza w Poznaniu, Seria Geografia 31.

Poniży L.: *Wpływ jakości środowiska przyrodniczego miasta na nasze zdrowie. Analiza przestrzenna na przykładzie Poznania*. 2008. Poznań: SORUS.

Wilson P.W.F., D'Agostino R.B., Levy D., Belanger A.M., Silbershatz H., William B. Kannel W.B.: Prediction of coronary heart disease using risk factor categories. *Circulation* 1998; 97:1837–1847.

Rona R.J.: The impact of the environment on height in Europe: conceptual and theoretical considerations *Ann Hum Biol* 2000; 27(2):111– 126.

Tanner J.M.: Growth as a mirror of the condition of society: Secular trends and class distinctions. In: *Human Growth. A Multidisciplinary Review*. A. Demirjian (Ed.) 1986:3–34. London: Taylor and Francis.

World Health Organization. Social determinants of health and health equity: "the causes of the causes". In: *The interim statement of the Commission on social determinants of health* 2007. Available at www.who.int/social_determinants/.../interim_statement/en/index.html Accessed March 1, 2011.

World Bank. *Environmental Health and Child Survival: Epidemiology, Economics, Experiences*. Environment and Development Series. 2008. Washington, DC: World Bank; Available at http://www.worldcat.org/title/environmental-health-and-child-survival-epidemiology-economics-experiences

# Physical Growth and Fitness

Maria Kaczmarek, Joachim Cieślik, Tomasz Hanć,
Magdalena Durda, Magdalena Skrzypczak

# Characteristics of adolescent physical growth – results of the ADOPOLNOR project

**Abstract:** This paper aims to present anthropometric growth characteristics based on cross-sectional studies on children and adolescents aged 10–18. A total of 4,828 individuals (2,445 girls and 2,383 boys) from the region of Wielkopolska were examined under the project. The tests covered 20 somatic traits and the analysis of body composition based on the impedance method. Body composition measurements were made with a BIA 101 analyser and Bodygram® software. Body Mass Index (BMI) was also calculated. With regard to three parameters, i.e. height, weight and BMI, the studied group was compared with the standards set for the city of Poznań and, additionally, with those proposed WHO. As the most important finding of the comparison, body weight of boys in the studied group was shown to be statistically significantly bigger than the Poznań standard for all analysed age groups, the mean difference being 2.27 kg. The comparison of BMIs revealed statistically significant differences only for the ages of 10, 13 and 14. The difference between the compared groups was in the range of 0.4–0.7 kg/m$^2$. The girls under study also had higher BMI levels (the difference ranging from 0.48 to 0.89 kg/m$^2$) for the ages of 10, 16, 17 and 18 years. The results reflect the global tendency for children and adolescents to develop overweight and obesity, which in the case of the studied sample related to changes of the socio-economic environment driven by the economic system transformation.

**Key words:** physical growth, anthropometric traits, growth standards, environmental factors

## Introduction

Physical growth in the process of ontogeny is understood as quantitative change involving the enlargement of body weight and size as well as the shaping of its proportions. All things considered, however, body growth is a manifestation of external effects of the ontogenetic process, such as differentiation and maturation of the organism [Cieślik et al. 1985]. The self-regulatory nature of the physical growth process is reflected in the specific interaction that occurs between the genetic

make-up of a generation and the environment where that growth takes place [Wolański 2005]. This means that genes and the external environment are both responsible for the quality of human physical growth. The proportions of either factor in the process are variable, depending on a generation's genotypic value and the ever changing quality of external environment factors. The physical development level of a given generation can, therefore, be seen as a reflection of that interdependence where quality of the process relies primarily on the intensity of the modifying impact of external environment factors [Cieślik 1980]. For that reason, physical growth characteristics are valuable indicators for assessing the quality of external environment.

The children and adolescents studied under the ADOPOLNOR project were born, brought up and physically shaped after 1988, that is after Poland's transition to the free-market economy, a major make-over from the previous regime. The new system brought about new components of the socio-economic environment. In this context, a question arises if this new, diverse and unstable configuration of socio-economic factors pertinent to the new economic system will prove to be a modifying agent for the gene pool of the analysed generation to the point of affecting its physical growth. To answer this question is one of the reasons behind the presentation of the results of the study on physical growth characteristics of said generation. We also want to find out if and to what degree the results of the ADOPOLNOR project differ from the physical growth levels set for the young generation of the city of Poznań that became adult (18 years old) in 2000 [Krawczyński et al. 2000] and relative standards proposed by the World Health Organisation [WHO 2006].

## Materials and methods

The study employed anthropometric data concerning children and adolescents aged 10–18. It was cross-sectional in nature and was conducted at primary, lower secondary and upper-secondary schools of Wielkopolska. The investigation comprised a total of 4,828 individuals, including 2,445 and 2,383 boys (Table 1). The measurements were taken by a team of anthropologists from the Department of Human Developmental Biology, Adam Mickiewicz University, Poznań, in cooperation with specially trained university students and school nurses (the training concerned techniques of measuring particular body parameters). The tests covered 20 somatic traits and the analysis of body composition. For detailed list of measurements see Table 2. BMI levels were also established based on height and weight data.

Table 1. Sample size

| | Boys | Girls | Total |
|---|---|---|---|
| Anthropometric data | 2383 | 2445 | 4828 |
| Body composition | 903 | 1041 | 1944 |

The anthropometric examinations were made upon prior approval of the subjects, their parents or other legal caretakers. All measurements were performed on the right side of the body using standard techniques by means of

Table 2. List of measurements taken in the survey

| Type of measurement | Measurement |
|---|---|
| Length/height measurements | Body height (B-v) |
| | Lower extremity length (B-sy) |
| | Upper extremity length (a-da) |
| | Trunk length (sst-sy) |
| | Head and neck length (v-sst) |
| Width/diameter measurements | Chest width (thl-thl) |
| | Chest depth (xi-thl) |
| | Shoulder width (a-a) |
| | Hip width (ic-ic) |
| Circumferences | Chest circumference |
| | Waist circumference |
| | Hip circumeference |
| | Arm circumference |
| | Forearm circumference |
| | Thigh circumference |
| | Shin circumference |
| Skinfolds | Triceps skinfold |
| | Subscapular skinfold |
| | Abdominal skinfold |
| Body composition | Body fat mass |
| | Body fat free mass |
| | Body muscle mass |
| | Body cell mass |
| | Total body water |
| | Body intracellular water |
| | Body extracellular water |
| | Body mass |

GMP anthropometers (measurement precision=1 mm), electronic scales (measurement precision=100 g) and GMP fat testers (measurement precision=0.2 mm). Tets were taken at school nursery rooms. Before examination, subjects were asked to undress to underwear.

Body composition was analysed in 1,944 individuals (903 boys and 1,041 girls). The evaluation was made with an impedance analyser by AKERN (BIA 101, voltage=50 KHz, current=800 mA). Impedance is defined as a function of fat-free tissue resistivity of body cross-section and length [Lewitt et al. 2007] and can be calculated using the following formula:

$$Z = ro \times L\text{^}2 \div a \times L \quad (1)$$

where:

Z – impedance (Ω)
ro – resistivity (Ω/cm)
L – body height (Ω/cm)
a – body cross-section ($cm^2$)

The examination was made with skin electrodes connected to the right side of the body. Prior to examination, in order to ensure even distribution of liquids, subjects were requested to lie down in a horizontal position for 5 minutes. They had also been instructed not to eat or drink anything for 3 hours preceding the test and to avoid physical effort for 6 hours preceding the test. Individuals with cardio-stimulators or other implanted controllers, e.g medicine applicators, were disqualified from the study.

Bodygram® Pro software was applied to analyse body composition based on input values of body resistance and reactance, height and weight. The study used percentage values obtained for particular body parameters

The data were classified into age groups divided by yearly intervals where one group encompassed, for example, individuals aged from 9.50 to 10.49 with mean age of 10.00. Detailed figures for particular age categories, mean values, medians and standard deviations for each parameter were shown in a tabular form separately for girls and boys.

In order to evaluate studied individuals' growth level versus current standard for Wielkopolska, the obtained results were compared with the standards set for the city of Poznań as published by Krawczyński, Krzyżaniak and Walkowiak in 2000. The significance of differences between mean height, weight and BMI levels in particular age categories were assessed with the *t-Student* test. The charts (Fig. 1–6) provide additional graphic comparison of weight, height and BMI levels with standards set for Poznań and those developed by the World Health Organisation [WHO 2006]. As WHO does not state body weight medians for the age categories dealt with in this study, the charts present weight values of the ADOPOLNOR sample only in relation to equivalent Poznań population. The medians in the charts were smoothed with the LMS method [Cole 1990, Cole and Green 1992].

The list of anthropometric traits and body composition tests is given in Table 2.

# Results

## Physical growth characteristics for children and adolescents from Wielkopolska region

Results of the study are presented in tables containing statistical characteristics of physical growth for children and adolescents from Wielkopolska region examined in the period of ontogeny, from 10 to 18 years of age. Mean values, medians and standard deviations here included show the level and variability of the sample

group's physical development. The number of subjects in particular age groups, as well as relative mean values and medians (their mutual relations), are in turn illustrative of the reliability of the sample, being well representative of the children and adolescent population of Wielkopolska.

The obtained physical growth parameters form a model reflection of the studied generation's ontogenetic process. The analysed traits are of different diagnostic values in the evaluation of the process. For that reason, our results are presented in three selected groups of variables that refer to qualitatively different aspects of the studied growth process. The sections below present diagnostic indicators used for physical growth evaluation, that is weight, height and BMI (Table 3–5), body proportion parameters taking account of the remaining measurements (Table 6–23), and body composition data (Table 24–30).

## Characteristics of physical growth

Table 3. Stature (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 142.40 | 142.00 | 5.84 | 148 | 142.99 | 142.35 | 6.74 |
| 11 | 269 | 146.93 | 146.70 | 7.43 | 265 | 147.42 | 147.20 | 7.31 |
| 12 | 240 | 153.19 | 153.00 | 7.44 | 285 | 152.57 | 153.20 | 7.94 |
| 13 | 227 | 158.71 | 158.20 | 8.62 | 276 | 158.83 | 158.95 | 6.72 |
| 14 | 263 | 166.89 | 167.40 | 8.94 | 248 | 162.29 | 162.30 | 6.08 |
| 15 | 262 | 172.19 | 172.50 | 7.41 | 300 | 163.55 | 164.00 | 5.76 |
| 16 | 252 | 175.66 | 175.95 | 7.23 | 311 | 164.20 | 164.00 | 5.86 |
| 17 | 273 | 177.55 | 177.40 | 6.62 | 213 | 164.92 | 165.40 | 6.01 |
| 18 | 304 | 178.27 | 178.05 | 6.63 | 290 | 164.15 | 163.95 | 5.74 |

Table 4. Body mass (kg)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 37.28 | 34.75 | 8.42 | 148 | 37.49 | 36.50 | 8.34 |
| 11 | 269 | 40.38 | 38.00 | 9.74 | 265 | 39.37 | 37.40 | 9.58 |
| 12 | 240 | 45.16 | 43.00 | 11.66 | 285 | 43.96 | 43.10 | 10.50 |
| 13 | 227 | 50.13 | 48.50 | 12.91 | 276 | 48.45 | 48.00 | 9.39 |
| 14 | 263 | 57.16 | 55.00 | 14.05 | 248 | 53.54 | 52.50 | 10.04 |
| 15 | 262 | 60.43 | 58.65 | 12.14 | 300 | 54.33 | 53.00 | 10.24 |
| 16 | 252 | 66.28 | 65.00 | 11.69 | 311 | 57.54 | 56.40 | 9.03 |
| 17 | 273 | 69.40 | 68.00 | 11.63 | 213 | 59.21 | 57.00 | 10.25 |
| 18 | 304 | 71.08 | 69.50 | 11.76 | 290 | 58.08 | 57.00 | 9.51 |

Table 5. BMI (kg/m$^2$)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 18.40 | 17.68 | 3.28 | 148 | 18.29 | 17.83 | 3.29 |
| 11 | 269 | 18.52 | 17.75 | 3.36 | 265 | 18.01 | 17.34 | 3.26 |
| 12 | 240 | 19.14 | 18.11 | 3.68 | 285 | 18.73 | 18.13 | 3.43 |
| 13 | 227 | 19.81 | 18.76 | 3.96 | 276 | 19.21 | 18.76 | 3.11 |
| 14 | 263 | 20.41 | 19.54 | 3.89 | 248 | 20.31 | 19.76 | 3.15 |
| 15 | 262 | 20.37 | 19.57 | 3.42 | 300 | 20.27 | 19.77 | 3.32 |
| 16 | 252 | 21.37 | 20.87 | 3.17 | 311 | 21.28 | 20.76 | 3.08 |
| 17 | 273 | 21.93 | 21.55 | 3.05 | 213 | 21.63 | 20.92 | 3.40 |
| 18 | 304 | 22.31 | 21.88 | 3.22 | 290 | 21.45 | 21.09 | 3.08 |

## Body proportion

Table 6. Lower extremity length [B-sy] (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 72.05 | 72.00 | 4.30 | 148 | 73.94 | 73.60 | 4.33 |
| 11 | 269 | 75.33 | 75.30 | 4.84 | 265 | 76.62 | 76.30 | 4.85 |
| 12 | 240 | 79.18 | 79.70 | 4.58 | 285 | 79.47 | 79.60 | 4.92 |
| 13 | 227 | 82.27 | 82.10 | 5.16 | 276 | 82.44 | 82.10 | 4.51 |
| 14 | 263 | 86.44 | 86.40 | 5.53 | 248 | 84.03 | 83.65 | 4.13 |
| 15 | 262 | 89.51 | 89.60 | 4.54 | 300 | 84.44 | 84.60 | 4.44 |
| 16 | 252 | 91.06 | 90.95 | 4.61 | 311 | 84.37 | 84.20 | 4.42 |
| 17 | 273 | 91.58 | 91.00 | 4.50 | 213 | 84.47 | 84.70 | 4.51 |
| 18 | 304 | 91.65 | 91.50 | 5.13 | 290 | 84.35 | 84.25 | 4.21 |

Table 7. Upper extremity length [a-da] (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 62.94 | 62.80 | 3.20 | 148 | 62.63 | 62.40 | 3.47 |
| 11 | 269 | 65.12 | 65.10 | 3.85 | 265 | 64.49 | 64.50 | 4.00 |
| 12 | 240 | 67.73 | 67.50 | 4.36 | 285 | 66.74 | 66.80 | 3.84 |
| 13 | 227 | 70.42 | 70.10 | 4.50 | 276 | 69.71 | 69.45 | 3.86 |
| 14 | 263 | 74.38 | 74.90 | 4.74 | 248 | 71.19 | 71.20 | 3.64 |
| 15 | 262 | 76.59 | 76.65 | 4.07 | 300 | 71.66 | 71.90 | 3.58 |
| 16 | 252 | 78.46 | 78.50 | 4.25 | 311 | 71.99 | 72.40 | 3.76 |
| 17 | 273 | 79.00 | 78.80 | 4.22 | 213 | 71.54 | 71.50 | 3.75 |
| 18 | 304 | 78.98 | 79.20 | 4.19 | 290 | 71.38 | 71.60 | 3.31 |

Table 8. Trunk length [sst-sy] (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 42.23 | 42.10 | 2.96 | 148 | 41.21 | 41.40 | 2.90 |
| 11 | 269 | 43.06 | 43.00 | 3.21 | 265 | 42.46 | 42.40 | 3.17 |
| 12 | 240 | 45.04 | 45.10 | 3.95 | 285 | 44.28 | 44.30 | 3.71 |
| 13 | 227 | 46.15 | 46.20 | 4.01 | 276 | 45.99 | 45.95 | 3.21 |
| 14 | 263 | 48.92 | 48.90 | 4.41 | 248 | 47.76 | 47.80 | 2.90 |
| 15 | 262 | 50.54 | 50.70 | 3.78 | 300 | 48.47 | 48.50 | 2.90 |
| 16 | 252 | 51.74 | 51.60 | 3.66 | 311 | 48.84 | 48.90 | 3.03 |
| 17 | 273 | 53.25 | 53.10 | 3.75 | 213 | 49.67 | 49.80 | 2.99 |
| 18 | 304 | 53.73 | 53.70 | 3.64 | 290 | 49.21 | 49.30 | 3.14 |

Table 9. Head and neck length [v-sst] (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 28.13 | 28.00 | 1.75 | 148 | 27.85 | 27.70 | 1.44 |
| 11 | 269 | 28.54 | 28.60 | 1.76 | 265 | 28.34 | 28.40 | 1.76 |
| 12 | 240 | 28.97 | 29.00 | 1.87 | 285 | 28.82 | 29.00 | 1.56 |
| 13 | 227 | 30.29 | 29.90 | 2.84 | 276 | 30.40 | 30.00 | 2.47 |
| 14 | 263 | 31.54 | 31.20 | 2.53 | 248 | 30.49 | 30.40 | 1.91 |
| 15 | 262 | 32.14 | 32.00 | 2.27 | 300 | 30.64 | 30.50 | 1.87 |
| 16 | 252 | 32.87 | 32.90 | 2.12 | 311 | 30.99 | 30.90 | 1.69 |
| 17 | 273 | 32.72 | 32.80 | 1.92 | 213 | 30.79 | 30.90 | 1.59 |
| 18 | 304 | 32.88 | 32.80 | 1.81 | 290 | 30.59 | 30.50 | 1.58 |

Table 10. Chest width [thl-thl] (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 22.01 | 21.40 | 2.43 | 148 | 21.65 | 21.35 | 2.30 |
| 11 | 269 | 22.22 | 22.00 | 2.21 | 265 | 21.84 | 21.70 | 2.36 |
| 12 | 240 | 23.07 | 22.80 | 2.26 | 285 | 22.64 | 22.50 | 2.20 |
| 13 | 227 | 23.94 | 23.70 | 2.34 | 276 | 23.53 | 23.20 | 2.20 |
| 14 | 263 | 25.04 | 24.70 | 2.39 | 248 | 24.31 | 24.10 | 1.94 |
| 15 | 262 | 26.09 | 26.00 | 2.26 | 300 | 24.59 | 24.30 | 1.85 |
| 16 | 252 | 27.12 | 27.00 | 2.41 | 311 | 25.14 | 25.00 | 1.92 |
| 17 | 273 | 27.81 | 27.80 | 2.22 | 213 | 25.32 | 25.00 | 2.20 |
| 18 | 304 | 28.39 | 28.20 | 2.29 | 290 | 24.98 | 25.00 | 1.98 |

Table 11. Chest depth [xi-thl] (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 15.83 | 15.20 | 2.20 | 148 | 15.80 | 15.60 | 2.36 |
| 11 | 269 | 15.94 | 15.50 | 1.99 | 265 | 15.82 | 15.40 | 2.32 |
| 12 | 240 | 16.63 | 16.10 | 2.14 | 285 | 16.46 | 16.20 | 2.21 |
| 13 | 227 | 17.15 | 16.80 | 2.45 | 276 | 17.02 | 16.80 | 2.04 |
| 14 | 263 | 18.06 | 17.80 | 2.41 | 248 | 17.51 | 17.30 | 2.10 |
| 15 | 262 | 18.30 | 18.00 | 2.09 | 300 | 17.44 | 17.30 | 2.22 |
| 16 | 252 | 19.04 | 18.60 | 2.01 | 311 | 18.03 | 17.90 | 1.97 |
| 17 | 273 | 19.28 | 19.00 | 2.15 | 213 | 18.13 | 18.00 | 2.23 |
| 18 | 304 | 19.55 | 19.35 | 2.30 | 290 | 17.88 | 17.80 | 1.85 |

Table 12. Shoulder width [a-a] (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 30.96 | 30.60 | 2.20 | 148 | 30.88 | 31.00 | 1.90 |
| 11 | 269 | 31.71 | 31.60 | 2.01 | 265 | 31.57 | 31.50 | 2.11 |
| 12 | 240 | 32.94 | 32.80 | 2.37 | 285 | 32.44 | 32.40 | 2.13 |
| 13 | 227 | 34.23 | 34.00 | 2.37 | 276 | 33.64 | 33.70 | 2.08 |
| 14 | 263 | 35.93 | 36.00 | 2.56 | 248 | 34.26 | 34.30 | 1.77 |
| 15 | 262 | 37.16 | 37.00 | 2.37 | 300 | 34.69 | 34.80 | 1.77 |
| 16 | 252 | 38.68 | 38.60 | 2.46 | 311 | 35.09 | 35.00 | 1.90 |
| 17 | 273 | 39.63 | 39.50 | 2.14 | 213 | 35.41 | 35.40 | 1.90 |
| 18 | 304 | 39.99 | 40.00 | 2.46 | 290 | 35.23 | 35.20 | 1.97 |

Table 13. Hip width [ic-ic] (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 22.84 | 22.40 | 2.26 | 148 | 23.35 | 23.00 | 2.54 |
| 11 | 269 | 23.51 | 23.10 | 2.37 | 265 | 23.40 | 23.10 | 2.25 |
| 12 | 240 | 24.43 | 24.00 | 2.36 | 285 | 24.35 | 24.20 | 2.21 |
| 13 | 227 | 24.97 | 24.70 | 2.40 | 276 | 25.24 | 25.05 | 2.06 |
| 14 | 263 | 26.23 | 26.00 | 2.65 | 248 | 26.04 | 26.20 | 2.20 |
| 15 | 262 | 26.74 | 26.80 | 2.05 | 300 | 26.59 | 26.50 | 1.89 |
| 16 | 252 | 27.80 | 27.60 | 2.23 | 311 | 27.35 | 27.20 | 1.94 |
| 17 | 273 | 28.41 | 28.40 | 2.03 | 213 | 27.80 | 27.70 | 1.80 |
| 18 | 304 | 28.45 | 28.40 | 2.08 | 290 | 27.53 | 27.30 | 1.80 |

Table 14. Chest circumference (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | Median | SD | N | Mean | Median | SD | N |
| 10 | 147 | 71.11 | 69.00 | 7.41 | 148 | 70.41 | 70.00 | 7.53 |
| 11 | 269 | 72.90 | 71.00 | 7.82 | 265 | 71.46 | 70.40 | 7.47 |
| 12 | 240 | 75.90 | 74.00 | 8.73 | 285 | 75.37 | 75.00 | 8.13 |
| 13 | 227 | 78.74 | 77.00 | 8.59 | 276 | 78.34 | 78.00 | 6.81 |
| 14 | 263 | 82.60 | 81.95 | 8.78 | 248 | 81.65 | 81.50 | 7.02 |
| 15 | 262 | 84.99 | 84.50 | 7.84 | 300 | 82.67 | 82.00 | 6.56 |
| 16 | 252 | 87.97 | 88.00 | 7.68 | 311 | 83.72 | 83.00 | 5.89 |
| 17 | 273 | 90.12 | 90.00 | 7.31 | 213 | 84.66 | 84.00 | 6.43 |
| 18 | 304 | 91.64 | 91.00 | 7.48 | 290 | 83.87 | 84.00 | 6.36 |

Table 15. Waist circumference (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 63.32 | 61.10 | 7.65 | 148 | 62.30 | 61.50 | 8.09 |
| 11 | 269 | 65.22 | 63.40 | 8.88 | 265 | 62.07 | 60.00 | 8.08 |
| 12 | 240 | 67.98 | 65.53 | 9.76 | 285 | 64.33 | 63.00 | 7.88 |
| 13 | 227 | 69.17 | 66.50 | 9.63 | 276 | 65.18 | 63.70 | 7.06 |
| 14 | 263 | 71.45 | 69.00 | 9.43 | 248 | 67.16 | 66.00 | 7.02 |
| 15 | 262 | 71.63 | 70.00 | 7.96 | 300 | 67.09 | 65.50 | 7.51 |
| 16 | 252 | 74.10 | 73.00 | 8.41 | 311 | 68.53 | 67.00 | 7.25 |
| 17 | 273 | 75.51 | 74.00 | 7.56 | 213 | 69.64 | 68.00 | 7.74 |
| 18 | 304 | 76.76 | 76.00 | 8.04 | 290 | 68.60 | 68.00 | 6.77 |

Table 16. Hip circumeference (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 75.22 | 74.00 | 7.26 | 148 | 76.29 | 76.43 | 7.64 |
| 11 | 269 | 76.91 | 75.65 | 8.99 | 265 | 77.05 | 76.00 | 8.85 |
| 12 | 240 | 80.42 | 79.00 | 8.71 | 285 | 81.02 | 81.00 | 8.72 |
| 13 | 227 | 82.96 | 81.40 | 9.33 | 276 | 85.19 | 85.10 | 8.04 |
| 14 | 263 | 87.67 | 86.90 | 9.50 | 248 | 89.08 | 88.95 | 8.02 |
| 15 | 262 | 89.34 | 88.39 | 8.06 | 300 | 90.41 | 89.18 | 7.55 |
| 16 | 252 | 91.59 | 92.00 | 8.92 | 311 | 91.06 | 92.00 | 8.40 |
| 17 | 273 | 92.25 | 92.50 | 9.32 | 213 | 93.24 | 93.00 | 7.83 |
| 18 | 304 | 93.07 | 93.10 | 8.95 | 290 | 92.92 | 93.00 | 8.51 |

Table 17. Arm circumference (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 21.97 | 21.35 | 3.40 | 148 | 21.87 | 21.50 | 3.09 |
| 11 | 269 | 22.17 | 21.92 | 3.51 | 265 | 21.70 | 21.00 | 3.23 |
| 12 | 240 | 23.19 | 22.50 | 3.52 | 285 | 22.77 | 22.00 | 3.41 |
| 13 | 227 | 23.81 | 23.50 | 3.82 | 276 | 23.65 | 23.50 | 3.10 |
| 14 | 263 | 24.57 | 24.00 | 3.60 | 248 | 24.08 | 23.50 | 3.12 |
| 15 | 262 | 25.28 | 25.00 | 3.31 | 300 | 24.22 | 24.00 | 3.24 |
| 16 | 252 | 26.02 | 26.00 | 3.52 | 311 | 24.63 | 24.50 | 3.33 |
| 17 | 273 | 26.50 | 26.00 | 3.10 | 213 | 25.37 | 25.30 | 3.05 |
| 18 | 304 | 27.49 | 27.00 | 3.51 | 290 | 25.29 | 25.00 | 3.13 |

Table 18. Forearm circumference (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 19.78 | 19.45 | 2.17 | 148 | 19.56 | 19.50 | 2.16 |
| 11 | 269 | 20.24 | 20.35 | 2.42 | 265 | 19.60 | 19.50 | 2.53 |
| 12 | 240 | 21.16 | 21.00 | 2.75 | 285 | 20.52 | 20.32 | 2.39 |
| 13 | 227 | 21.51 | 21.50 | 2.72 | 276 | 20.84 | 21.00 | 2.35 |
| 14 | 263 | 22.62 | 22.50 | 2.46 | 248 | 21.24 | 21.00 | 2.17 |
| 15 | 262 | 23.27 | 23.45 | 2.42 | 300 | 21.31 | 21.50 | 2.42 |
| 16 | 252 | 23.62 | 24.00 | 3.02 | 311 | 21.36 | 21.60 | 2.80 |
| 17 | 273 | 23.99 | 24.50 | 3.10 | 213 | 21.88 | 22.00 | 2.30 |
| 18 | 304 | 24.31 | 24.50 | 3.18 | 290 | 21.73 | 22.00 | 2.32 |

Table 19. Thigh circumference (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 42.66 | 41.35 | 6.34 | 148 | 43.86 | 43.60 | 5.57 |
| 11 | 269 | 44.37 | 43.19 | 6.43 | 265 | 44.42 | 43.18 | 6.26 |
| 12 | 240 | 46.68 | 45.50 | 6.44 | 285 | 47.06 | 46.50 | 6.65 |
| 13 | 227 | 47.72 | 47.00 | 6.66 | 276 | 49.16 | 49.00 | 5.95 |
| 14 | 263 | 49.69 | 49.00 | 6.52 | 248 | 50.82 | 50.65 | 5.79 |
| 15 | 262 | 50.27 | 50.00 | 6.04 | 300 | 51.60 | 51.50 | 5.67 |
| 16 | 252 | 51.23 | 51.00 | 5.98 | 311 | 51.86 | 52.00 | 6.17 |
| 17 | 273 | 52.07 | 51.65 | 6.25 | 213 | 53.38 | 53.00 | 5.87 |
| 18 | 304 | 52.86 | 53.00 | 5.87 | 290 | 53.28 | 54.00 | 5.96 |

Table 20. Shin circumference (cm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 29.55 | 28.50 | 3.18 | 148 | 29.81 | 29.85 | 3.31 |
| 11 | 269 | 30.61 | 30.00 | 3.83 | 265 | 30.09 | 29.50 | 3.79 |
| 12 | 240 | 31.91 | 31.50 | 4.25 | 285 | 31.45 | 31.00 | 3.92 |
| 13 | 227 | 32.76 | 32.50 | 4.00 | 276 | 32.37 | 32.50 | 3.55 |
| 14 | 263 | 34.19 | 34.00 | 4.14 | 248 | 33.66 | 33.50 | 3.61 |
| 15 | 262 | 34.65 | 34.54 | 3.45 | 300 | 33.82 | 34.00 | 3.24 |
| 16 | 252 | 35.01 | 35.00 | 4.30 | 311 | 34.16 | 34.50 | 3.82 |
| 17 | 273 | 35.43 | 36.00 | 4.79 | 213 | 34.80 | 35.00 | 3.73 |
| 18 | 304 | 35.67 | 36.40 | 4.38 | 290 | 34.66 | 35.00 | 4.00 |

Table 21. Triceps skinfold (mm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 12.88 | 11.20 | 6.12 | 148 | 14.31 | 13.60 | 5.47 |
| 11 | 269 | 12.85 | 11.40 | 5.93 | 265 | 14.08 | 13.20 | 5.29 |
| 12 | 240 | 13.45 | 11.40 | 6.50 | 285 | 14.40 | 13.00 | 6.10 |
| 13 | 227 | 12.57 | 11.00 | 6.23 | 276 | 14.27 | 13.90 | 5.09 |
| 14 | 263 | 11.86 | 9.80 | 6.44 | 248 | 15.38 | 14.80 | 5.10 |
| 15 | 262 | 10.75 | 9.00 | 5.58 | 300 | 16.10 | 15.40 | 5.65 |
| 16 | 252 | 11.16 | 8.80 | 6.62 | 311 | 16.71 | 16.00 | 5.56 |
| 17 | 273 | 10.64 | 9.40 | 4.90 | 213 | 17.58 | 16.80 | 5.71 |
| 18 | 304 | 11.05 | 10.00 | 5.29 | 290 | 17.30 | 17.00 | 5.69 |

Table 22. Subscapular skinfold (mm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 10.66 | 7.60 | 7.17 | 148 | 11.79 | 9.30 | 7.86 |
| 11 | 269 | 10.96 | 8.80 | 7.03 | 265 | 11.61 | 9.60 | 6.37 |
| 12 | 240 | 12.63 | 9.00 | 8.76 | 285 | 12.43 | 10.20 | 6.66 |
| 13 | 227 | 11.59 | 8.00 | 8.28 | 276 | 12.54 | 10.50 | 6.57 |
| 14 | 263 | 10.75 | 8.00 | 7.63 | 248 | 13.21 | 11.80 | 5.86 |
| 15 | 262 | 10.27 | 8.00 | 6.10 | 300 | 14.04 | 12.00 | 7.03 |
| 16 | 252 | 10.89 | 8.40 | 6.57 | 311 | 14.82 | 13.60 | 6.63 |
| 17 | 273 | 10.67 | 9.20 | 4.88 | 213 | 16.09 | 14.40 | 7.26 |
| 18 | 304 | 11.50 | 10.00 | 5.37 | 290 | 15.43 | 14.05 | 6.23 |

Table 23. Abdominal skinfold (mm)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 147 | 13.57 | 9.80 | 9.48 | 148 | 14.56 | 12.45 | 8.70 |
| 11 | 269 | 14.12 | 11.00 | 9.71 | 265 | 14.89 | 13.40 | 8.44 |
| i012 | 240 | 15.29 | 12.20 | 10.22 | 285 | 15.56 | 13.60 | 8.18 |
| 13 | 227 | 13.93 | 10.00 | 9.87 | 276 | 15.93 | 14.40 | 7.51 |
| 14 | 263 | 13.57 | 10.40 | 9.16 | 248 | 17.64 | 16.20 | 7.43 |
| 15 | 262 | 12.68 | 9.60 | 8.19 | 300 | 18.15 | 16.80 | 7.89 |
| 16 | 252 | 13.26 | 10.00 | 8.44 | 311 | 19.12 | 19.00 | 6.99 |
| 17 | 273 | 13.17 | 11.00 | 7.13 | 213 | 19.90 | 18.80 | 7.71 |
| 18 | 304 | 14.10 | 11.60 | 8.17 | 290 | 19.26 | 18.00 | 7.12 |

## Body composition

Table 24. Body fat mass (%)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 79 | 9.29 | 8.70 | 5.81 | 85 | 9.35 | 8.20 | 5.32 |
| 11 | 109 | 9.21 | 8.00 | 4.86 | 121 | 9.03 | 8.20 | 5.29 |
| 12 | 92 | 10.54 | 9.00 | 6.05 | 96 | 10.86 | 9.50 | 5.49 |
| 13 | 66 | 12.06 | 11.50 | 7.37 | 98 | 11.68 | 10.30 | 5.27 |
| 14 | 103 | 11.64 | 9.90 | 7.74 | 100 | 13.76 | 13.20 | 5.46 |
| 15 | 90 | 11.33 | 9.90 | 6.52 | 103 | 13.82 | 13.50 | 5.97 |
| 16 | 99 | 11.75 | 10.40 | 6.01 | 133 | 14.36 | 14.00 | 5.39 |
| 17 | 129 | 11.61 | 10.10 | 7.03 | 137 | 15.93 | 14.70 | 6.91 |
| 18 | 136 | 12.33 | 10.80 | 6.16 | 168 | 15.56 | 14.40 | 6.57 |

Table 25. Body fat free mass (%)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 79 | 28.51 | 28.00 | 4.56 | 85 | 28.68 | 28.15 | 5.90 |
| 11 | 109 | 30.79 | 30.30 | 5.77 | 121 | 30.87 | 29.40 | 6.36 |
| 12 | 92 | 35.58 | 34.75 | 7.53 | 96 | 34.90 | 34.25 | 7.90 |
| 13 | 66 | 41.78 | 41.50 | 10.35 | 98 | 37.90 | 37.40 | 6.29 |
| 14 | 103 | 47.31 | 46.10 | 9.24 | 100 | 41.79 | 40.60 | 8.24 |
| 15 | 90 | 50.44 | 49.80 | 9.22 | 103 | 41.10 | 39.50 | 6.58 |
| 16 | 99 | 54.53 | 54.50 | 9.53 | 133 | 43.38 | 42.65 | 5.77 |
| 17 | 129 | 58.15 | 57.70 | 7.69 | 137 | 43.15 | 42.40 | 5.07 |
| 18 | 136 | 60.40 | 60.05 | 7.13 | 168 | 42.88 | 42.35 | 4.67 |

Tabela 26. Body cell mass (% of weight)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 79 | 13,98 | 13,50 | 3,44 | 85 | 14,16 | 13,30 | 14,16 |
| 11 | 109 | 15,10 | 15,00 | 3,54 | 121 | 15,05 | 14,60 | 15,05 |
| 12 | 92 | 16,50 | 16,00 | 4,25 | 96 | 16,51 | 15,75 | 16,51 |
| 13 | 66 | 19,89 | 20,00 | 4,52 | 98 | 19,21 | 18,90 | 19,21 |
| 14 | 103 | 24,03 | 23,30 | 5,45 | 100 | 21,88 | 21,60 | 21,87 |
| 15 | 90 | 25,04 | 24,35 | 5,83 | 103 | 21,17 | 20,30 | 21,17 |
| 16 | 99 | 29,36 | 28,50 | 8,10 | 133 | 23,28 | 22,30 | 23,28 |
| 17 | 129 | 31,20 | 31,40 | 5,35 | 137 | 22,45 | 21,70 | 22,45 |
| 18 | 136 | 32,61 | 32,20 | 4,65 | 168 | 22,24 | 21,80 | 22,24 |

Table 27. Body muscle mass (%)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 79 | 17.38 | 16.70 | 4.09 | 85 | 17.50 | 16.60 | 4.72 |
| 11 | 109 | 18.75 | 18.60 | 4.30 | 121 | 18.61 | 18.20 | 4.03 |
| 12 | 92 | 20.51 | 19.80 | 5.15 | 96 | 20.47 | 19.55 | 5.82 |
| 13 | 66 | 24.67 | 24.80 | 5.47 | 98 | 23.75 | 23.35 | 4.98 |
| 14 | 103 | 29.83 | 29.00 | 6.61 | 100 | 26.97 | 26.80 | 5.23 |
| 15 | 90 | 31.03 | 30.05 | 7.08 | 103 | 26.15 | 25.20 | 5.06 |
| 16 | 99 | 36.05 | 34.90 | 9.49 | 133 | 28.59 | 27.45 | 5.94 |
| 17 | 129 | 38.38 | 38.50 | 6.23 | 137 | 27.77 | 26.80 | 5.56 |
| 18 | 136 | 40.07 | 39.55 | 5.38 | 168 | 27.47 | 27.05 | 4.29 |

Table 28. Total body water (%)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 79 | 24.13 | 24.10 | 4.12 | 85 | 22.76 | 22.80 | 3.47 |
| 11 | 109 | 26.17 | 26.40 | 3.90 | 121 | 24.36 | 24.10 | 3.54 |
| 12 | 92 | 28.49 | 28.55 | 5.31 | 96 | 26.47 | 26.25 | 3.96 |
| 13 | 66 | 31.77 | 32.65 | 5.14 | 98 | 28.65 | 28.50 | 3.24 |
| 14 | 103 | 36.09 | 35.60 | 6.54 | 100 | 30.25 | 30.10 | 4.24 |
| 15 | 90 | 37.24 | 37.25 | 6.62 | 103 | 29.71 | 29.00 | 4.08 |
| 16 | 99 | 40.29 | 40.00 | 6.54 | 133 | 31.47 | 31.10 | 3.68 |
| 17 | 129 | 42.45 | 42.30 | 5.71 | 137 | 31.61 | 31.00 | 3.76 |
| 18 | 136 | 44.18 | 43.90 | 5.22 | 168 | 31.38 | 31.00 | 3.44 |

Table 29. Body extracellular water (%)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 79 | 9.88 | 9.70 | 1.68 | 85 | 9.51 | 9.40 | 1.67 |
| 11 | 109 | 10.45 | 10.10 | 1.94 | 121 | 10.22 | 9.90 | 1.87 |
| 12 | 92 | 11.84 | 11.65 | 2.58 | 96 | 11.41 | 11.10 | 2.37 |
| 13 | 66 | 13.90 | 14.40 | 2.78 | 98 | 12.88 | 12.60 | 2.20 |
| 14 | 103 | 16.92 | 16.60 | 3.16 | 100 | 14.09 | 13.75 | 2.67 |
| 15 | 90 | 17.55 | 17.35 | 3.25 | 103 | 13.96 | 13.40 | 2.69 |
| 16 | 99 | 18.19 | 17.90 | 3.24 | 133 | 14.57 | 14.20 | 2.75 |
| 17 | 129 | 19.49 | 19.30 | 2.99 | 137 | 15.27 | 14.90 | 2.85 |
| 18 | 136 | 20.11 | 19.90 | 3.37 | 168 | 14.90 | 14.95 | 2.29 |

Table 30. Body intracellular water (%)

| Age | Boys | | | | Girls | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Median | SD | N | Mean | Median | SD |
| 10 | 79 | 14.30 | 14.80 | 3.02 | 85 | 13.26 | 13.50 | 2.57 |
| 11 | 109 | 15.72 | 15.90 | 2.39 | 121 | 14.14 | 14.20 | 2.24 |
| 12 | 92 | 16.65 | 16.95 | 3.16 | 96 | 15.06 | 15.00 | 1.82 |
| 13 | 66 | 17.88 | 18.25 | 3.03 | 98 | 15.77 | 15.80 | 1.98 |
| 14 | 103 | 19.17 | 19.10 | 4.01 | 100 | 16.15 | 16.35 | 2.30 |
| 15 | 90 | 19.68 | 19.90 | 3.86 | 103 | 15.75 | 16.10 | 2.61 |
| 16 | 99 | 22.10 | 21.50 | 4.46 | 133 | 16.90 | 16.80 | 3.00 |
| 17 | 129 | 22.96 | 23.20 | 3.89 | 137 | 16.35 | 15.90 | 3.02 |
| 18 | 136 | 24.08 | 23.80 | 3.31 | 168 | 16.48 | 16.10 | 2.64 |

## Studied sample in comparison with Poznań and global growth standards

Tables 31–36 and Figures 1–6 provide a comparison of height, weight and BMI of children and teens from Wielkopolska region investigated under the ADOPOLNOR project with the most recent growth standards set for young population of Poznań [Krawczyński et al. 2000]. Median curves for WHO standards [2006] were also included in some of the charts as an additional reference.

Boys from the ADOPOLNOR sample were statistically significantly taller than the Poznań standard only for the age of 10 years (difference=1.60 cm, $p<0.05$). No statistically significant differences were found for the other age categories. Ten-year-old girls were also taller than the reference level. The difference (2.69 cm) was statistically significant at the level of 0.01. For the age group of 18 years, the *t-Student* test demonstrated an opposite tendency: the girls under study were sta-

Table 31. Comparison of means stature data of boys from the ADOPOLNOR survey with reference data of Poznań children and youth [Krawczyński et al. 2000]

| Age | ADOPOLNOR | | | Poznań 2000 | | | *t* |
|---|---|---|---|---|---|---|---|
| | N | Mean | SD | N | Mean | SD | |
| 10 | 147 | 142.40 | 5.84 | 1281 | 140.80 | 5.80 | 2.15* |
| 11 | 269 | 146.93 | 7.43 | 1494 | 146.60 | 6.10 | 0.95 |
| 12 | 240 | 153.19 | 7.44 | 1566 | 152.20 | 6.90 | 1.27 |
| 13 | 227 | 158.71 | 8.62 | 1524 | 158.80 | 7.90 | –0.79 |
| 14 | 263 | 166.89 | 8.94 | 1997 | 166.70 | 7.90 | –0.20 |
| 15 | 262 | 172.19 | 7.41 | 1588 | 171.40 | 7.30 | 0.93 |
| 16 | 252 | 175.66 | 7.23 | 1245 | 175.60 | 6.40 | 0.53 |
| 17 | 273 | 177.55 | 6.62 | 1124 | 177.50 | 6.10 | 0.31 |
| 18 | 304 | 178.27 | 6.63 | 1135 | 178.70 | 6.40 | –0.94 |

*statistically significant at $p<0.05$
**statistically significant at $p<0.01$

Table 32. Comparison of means stature data of girls from the ADOPOLNOR survey with reference data of Poznań children and youth [Krawczyński et al. 2000]

| ADOPOLNOR | | | | Poznań 2000 | | | |
|---|---|---|---|---|---|---|---|
| Age | N | Mean | SD | Age | N | Mean | SD |
| 10 | 148 | 142.99 | 6.74 | 1234 | 140.30 | 6.10 | 4.55** |
| 11 | 265 | 147.42 | 7.31 | 1575 | 147.30 | 6.80 | –0.31 |
| 12 | 285 | 152.57 | 7.94 | 1608 | 153.00 | 6.60 | –1.16 |
| 13 | 276 | 158.83 | 6.72 | 1462 | 159.00 | 5.90 | –1.24 |
| 14 | 248 | 162.29 | 6.08 | 2000 | 162.30 | 5.40 | –0.68 |
| 15 | 300 | 163.55 | 5.76 | 1444 | 164.10 | 5.40 | –1.52 |
| 16 | 311 | 164.20 | 5.86 | 1340 | 164.80 | 5.50 | –1.10 |
| 17 | 213 | 164.92 | 6.01 | 1359 | 165.90 | 5.40 | –1.45 |
| 18 | 290 | 164.15 | 5.74 | 1200 | 166.40 | 5.60 | –5.58** |

*statistically significant at $p<0.05$
**statistically significant at $p<0.01$

Tabela 33. Comparison of means body mass data of boys from the ADOPOLNOR survey with reference data of Poznań children and youth [Krawczyński et al. 2000]

| ADOPOLNOR | | | | Poznań 2000 | | | |
|---|---|---|---|---|---|---|---|
| Age | N | Mean | SD | Age | N | Mean | SD |
| 10 | 147 | 37.28 | 8.42 | 1281 | 34.30 | 6.10 | 5.44** |
| 11 | 269 | 40.38 | 9.74 | 1494 | 38.30 | 8.00 | 3.83** |
| 12 | 240 | 45.16 | 11.66 | 1566 | 42.60 | 8.50 | 4.19** |
| 13 | 227 | 50.13 | 12.91 | 1524 | 48.00 | 9.70 | 2.98** |
| 14 | 263 | 57.16 | 14.05 | 1997 | 54.80 | 9.70 | 3.55** |
| 15 | 262 | 60.43 | 12.14 | 1588 | 59.00 | 9.80 | 2.15* |
| 16 | 252 | 66.28 | 11.69 | 1245 | 63.80 | 9.40 | 3.79** |
| 17 | 273 | 69.40 | 11.63 | 1124 | 66.90 | 9.00 | 3.99** |
| 18 | 304 | 71.08 | 11.76 | 1135 | 69.10 | 9.10 | 3.29** |

*statistically significant at $p<0.05$
**statistically significant at $p<0.01$

Tabela 34. Comparison of means body mass data of girls from the ADOPOLNOR survey with reference data of Poznań children and youth [Krawczyński et al. 2000]

| ADOPOLNOR | | | | Poznań 2000 | | | |
|---|---|---|---|---|---|---|---|
| Age | N | Mean | SD | Age | N | Mean | SD |
| 10 | 148 | 37.49 | 8.34 | 1234 | 34.30 | 6.50 | 6.21** |
| 11 | 265 | 39.37 | 9.58 | 1575 | 38.30 | 7.60 | 1.88 |
| 12 | 285 | 43.96 | 10.50 | 1608 | 42.60 | 7.90 | 1.43 |
| 13 | 276 | 48.45 | 9.39 | 1462 | 48.00 | 7.70 | 0.87 |
| 14 | 248 | 53.54 | 10.04 | 2000 | 54.80 | 7.80 | 1.93 |
| 15 | 300 | 54.33 | 10.24 | 1444 | 59.00 | 7.30 | 0.47 |
| 16 | 311 | 57.54 | 9.03 | 1340 | 63.80 | 7.00 | 4.45** |
| 17 | 213 | 59.21 | 10.25 | 1359 | 66.90 | 6.70 | 5.23** |
| 18 | 290 | 58.08 | 9.51 | 1200 | 69.10 | 7.10 | 1.81 |

*statistically significant at $p<0.05$
**statistically significant at $p<0.01$

Tabela 35. Comparison of means BMI data of boys from the ADOPOLNOR survey with reference data of Poznań children and youth [Krawczyński et al. 2000]

| Age | ADOPOLNOR | | | Poznań 2000 | | | *t* |
|---|---|---|---|---|---|---|---|
| | N | Mean | SD | Age | N | Mean | |
| 10 | 147 | 18.40 | 3.28 | 1281 | 17.70 | 3.00 | 2.71** |
| 11 | 269 | 18.52 | 3.36 | 1494 | 18.30 | 3.30 | 1.02 |
| 12 | 240 | 19.14 | 3.68 | 1566 | 18.70 | 3.30 | 1.94 |
| 13 | 227 | 19.81 | 3.96 | 1524 | 19.30 | 3.40 | 2.08* |
| 14 | 263 | 20.41 | 3.89 | 1997 | 20.00 | 3.10 | 1.98* |
| 15 | 262 | 20.37 | 3.42 | 1588 | 20.40 | 3.20 | –0.16 |
| 16 | 252 | 21.37 | 3.17 | 1245 | 21.10 | 3.00 | 1.37 |
| 17 | 273 | 21.93 | 3.05 | 1124 | 21.60 | 3.00 | 1.71 |
| 18 | 304 | 22.31 | 3.22 | 1135 | 22.00 | 3.10 | 1.63 |

*statistically significant at $p<0.05$
**statistically significant at $p<0.01$

Tabela 36. Comparison of means BMI data of girls from the ADOPOLNOR survey with reference data of Poznań children and youth [Krawczyński et al. 2000]

| Age | ADOPOLNOR | | | Poznań 2000 | | | *t* |
|---|---|---|---|---|---|---|---|
| | N | Mean | SD | Age | N | Mean | |
| 10 | 148 | 18.29 | 3.29 | 1234 | 17.40 | 2.80 | 3.65** |
| 11 | 265 | 18.01 | 3.26 | 1575 | 18.10 | 3.20 | –0.44 |
| 12 | 285 | 18.73 | 3.43 | 1608 | 18.70 | 3.20 | 0.16 |
| 13 | 276 | 19.21 | 3.11 | 1462 | 19.50 | 3.30 | –1.39 |
| 14 | 248 | 20.31 | 3.15 | 2000 | 20.20 | 3.20 | 0.50 |
| 15 | 300 | 20.27 | 3.32 | 1444 | 20.40 | 2.90 | –0.72 |
| 16 | 311 | 21.28 | 3.08 | 1340 | 20.80 | 2.90 | 2.64** |
| 17 | 213 | 21.63 | 3.40 | 1359 | 20.90 | 2.80 | 3.53** |
| 18 | 290 | 21.45 | 3.08 | 1200 | 20.90 | 2.90 | 2.93** |

*statistically significant at $p<0.05$
**statistically significant at $p<0.01$

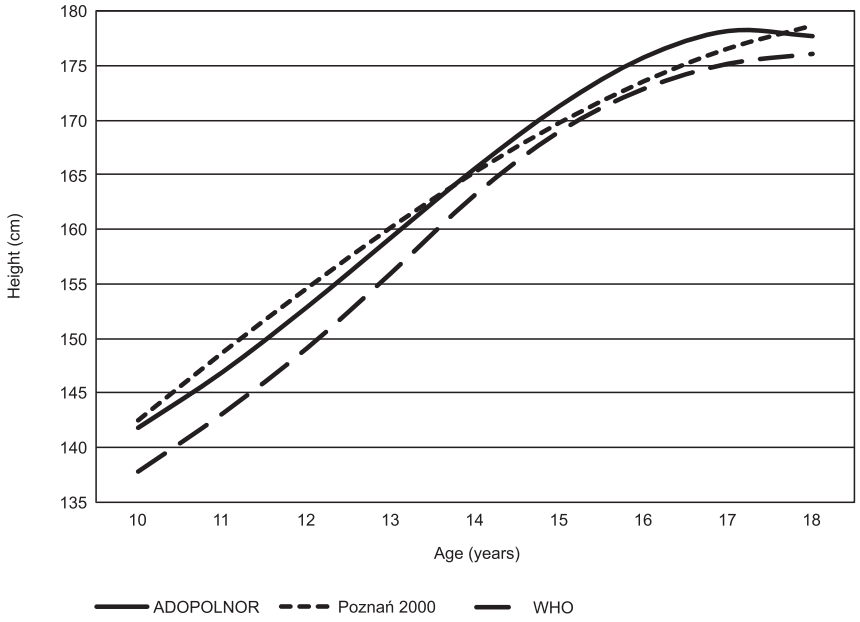



Fig. 1. Body height of boys from the ADOPOLNOR sample versus WHO growth standards [2006] and standards for children and adolescents from Poznań [Krawczyński et al. 2000] (the figure shows smoothed median curves)

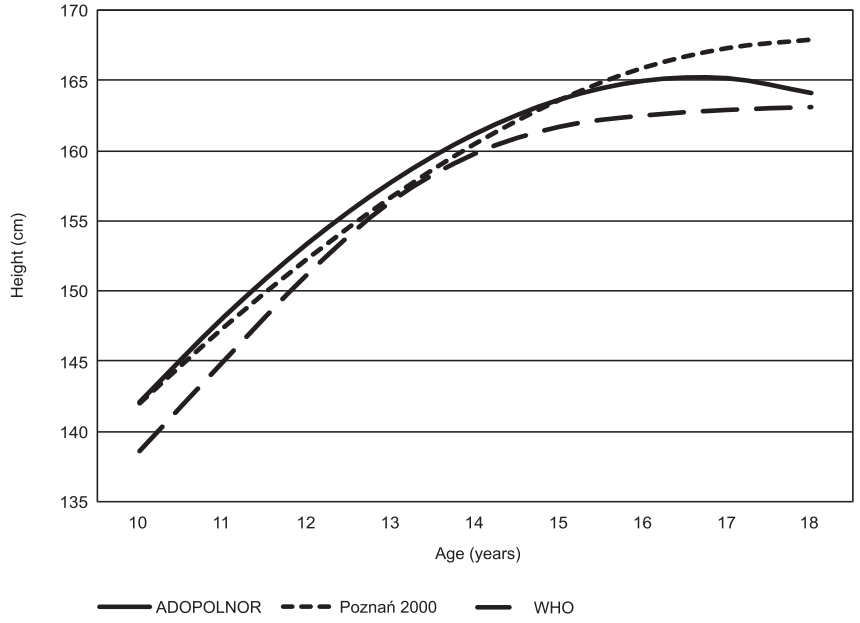



Fig. 2. Body height of girls from the ADOPOLNOR sample versus WHO growth standards [2006] and standards for children and adolescents from Poznań [Krawczyński et al. 2000] (the figure shows smoothed median curves)

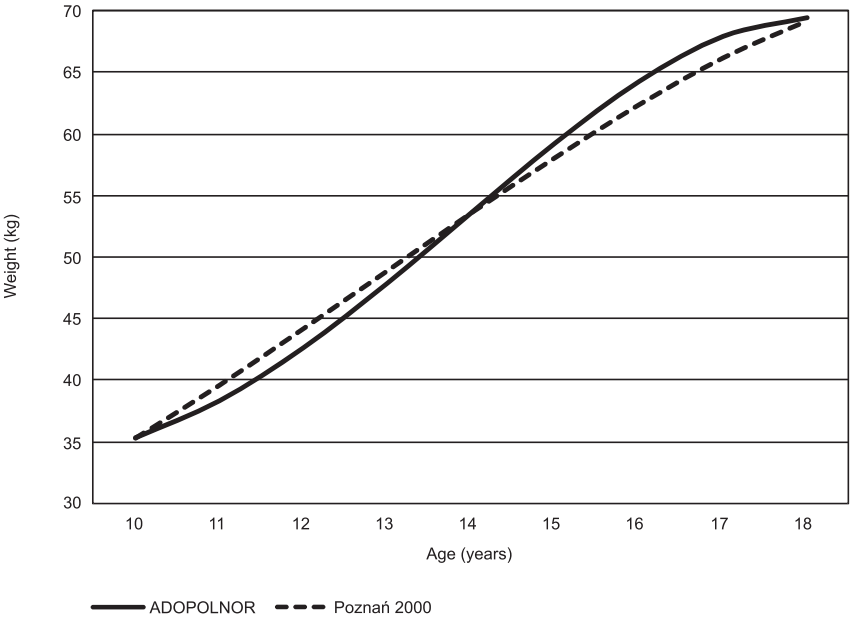



Fig. 3. Body weight of boys from the ADOPOLNOR sample versus standards for children and adolescents from Poznań [Krawczyński et al. 2000] (the figure shows smoothed median curves)

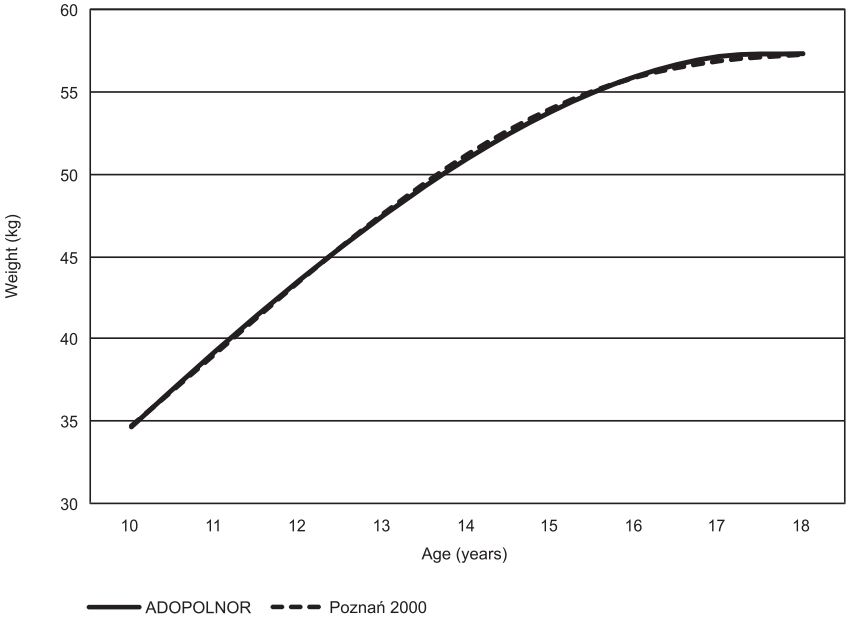



Fig. 4. Body weight of girls from the ADOPOLNOR sample versus standards for children and adolescents from Poznań [Krawczyński et al. 2000] (the figure shows smoothed median curves)

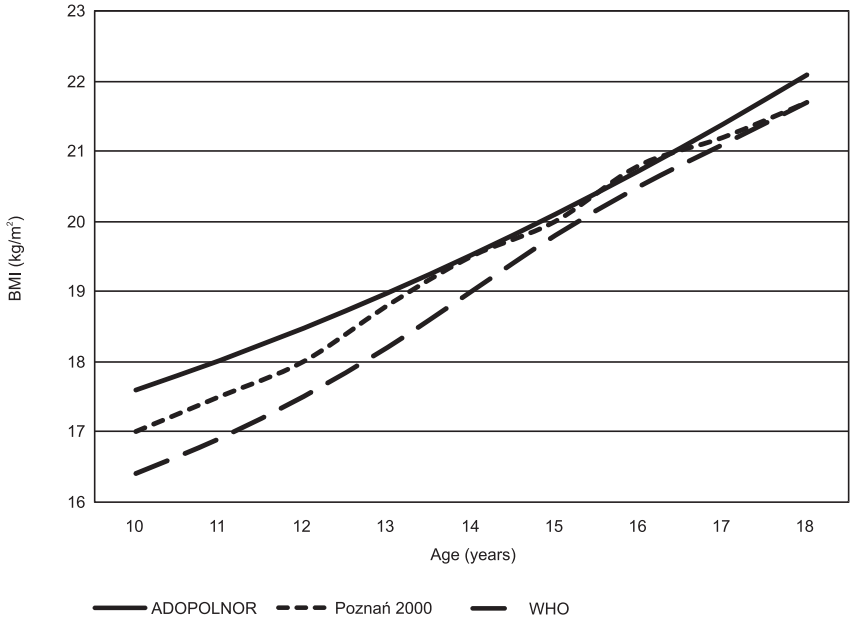



Fig. 5. BMI of boys from the ADOPOLNOR sample versus WHO growth standards [2006] and standards for children and adolescents from Poznań [Krawczyński et al. 2000] (the figure shows smoothed median curves)

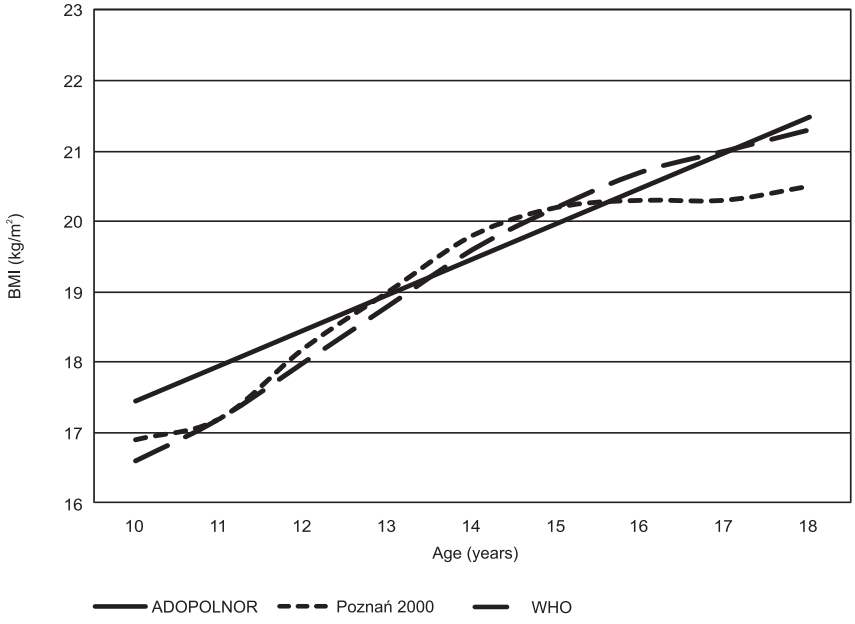



Fig. 6. BMI of girls from the ADOPOLNOR sample versus WHO growth standards [2007] and standards for children and adolescents from Poznań [Krawczyński et al. 2000] (the figure shows smoothed median curves)

tistically significantly shorter (difference=2.25 cm, $p<0.01$) than their counterparts referred to in the Poznań standards.

ADOPOLNOR boys' mean weight was statistically significantly larger than the Poznań standards for all examined age groups. A mean difference between analysed groups was 2.27 kg (min=1.43 kg; max=2.98 kg), with the largest disparity revealed by the *t-Student* for the age of 10 years. Girls' weight differed from the reference standard in three age groups: 10, 16 and 17 years. Ten-year-old ADOPOLNOR girls were found to have larger weight (difference=3.19 kg, $p<0.01$), while girls aged 16 (difference=6.26 kg, $p<0.01$) and 17 (difference=7.69 kg, $p<0.01$) had lighter weights than those set in the Poznań standard for adolescents.

Boys from the ADOPOLNOR sample showed a statistically significantly higher BMI versus the Poznań standards in three age categories: 10, 13, and 14 years. For the age of 10 years, the difference was 0.7 kg/m$^2$ ($p<0.01$), for the age of 13 years, 0.51 kg/m$^2$ ($p<0.05$), and for the age of 14 years, 0.41 kg/m$^2$ ($p<0.05$). No statistically significant differences were found for the other age categories. Girls showed a statistically significant difference ($p<0.05$) from the reference standard for the ages of 10, 16, 17 and 18 years. In those age groups, ADOPOLNOR girls presented with higher BMI levels than the Poznań standard. The difference was in the range of 0.48–0.89 kg/m$^2$.

## Discussion

The research methodology and the nature of results obtained determine the scope of discussion. The results clearly demonstrate that the examined physical development process is properly reflected by its characteristics which are in keeping to the genetic adaptive strategy for this period of ontogeny. The obtained levels of parameters analysed within this process are the outcome of the modifying activity of external environment factors [Cieślik 1985; Kaczmarek 1995; 2001].

As follows from the comparison of mean height, weight and BMI levels between the studied sample and the standards set for children and adolescents from the city of Poznań, new socio-economic conditions created by the free-market economic system have developed into a powerful environment capable of with modifying and shaping the examined growth features, although its implications are multi-directional. The new quality of the external environment and economic system, diverse and unstable as it was in the transitional stage of forming a new socio-economic configuration, have affected particular growth features in different ways.

Body height, in both boys and girls, does not differ significantly from the reference standard set for children and adolescents from Poznań in 2000 [Krawczyński et al. 2000]. Whereas body weight proved – unfortunately one should say – to be highly responsive and susceptible to the quality of the new environment [Cieślik 1980]. In the case of boys, mean differences in relation to the reference standard were found to be significant in all age groups. Girls proved to be more resistant to the influence of the new environment quality, with statisti-

cally significant disparities from reference levels recorded only for the ages of 10, 16 and 17. The weight measurement results for boys under study reflect, unfortunately, the globally observed tendency of overweight and obesity, which is more prominent in boys than girls [Cieślik and Mrowicka 2006]. Overall, this alarming increase in body weight, described in terms of mean values and presented as BMI expressed as a parameter relative to body height, turned out to be moderate for boys and part of girls. But both in boys and girls it also proved statistically significant in certain age groups.

The result section contains additional graphic comparisons of height, weight and BMI values with WHO standards using medians aligned with the LMS method. This places our growth results in another context, which we consider equally interesting.

Boys height in relation to Poznań standards [Krawczyński et al. 2000] and WHO standards [2006], are marked with the highest values for the ages of 14 and 18 years. Girls are characterised by higher height levels throughout the studied ontogenetic period as compared to both WHO and Poznań standards.

Median weight curve for the studied group in relation to the equivalent Poznań standards (no WHO data are available for this age range) shows a rising trend for boys from the age of 14 up to the age of 18. The median curve for the same feature in girls is the same throughout the whole ontogenetic period as that in the reference peer group.

BMI compared in the same way for boys shows higher values throughout the studied period. This reconfirms our concern expressed while analysing arithmetically derived weight characteristics. For girls, the BMI medians do not show the same tendency as in boys, being consistent with those set in WHO standards.

## Conclusion

The above presented study results and related comparisons with the current standards set for children and adolescents from the city of Poznań and those proposed by WHO allow the formulation of the following conclusions:

The biological evaluation of the studied group's physical growth levels constitutes a reflects perfectly the ontogenetic structure of the process, while the level of growth confirms a substantial contribution of external environment factors.

The socio-economic environment in the period when the studied children and adolescents were born and brought up proved to have had a modifying effect on their physical development features (height, weight and BMI).

The average level of physical growth in the studied group is characterised by higher values in comparison to both the generation of 2000 and, very importantly, the WHO-proposed standards.

## References

Cieślik J.: Wielopoziomowy rozwój fenotypowy osobnika i populacji w ontogenezie. Seria antropologiczna nr 7. 1980. Poznań: Wydawnictwo Naukowe UAM.

Cieślik J., Drozdowska M., Malinowski M.: Zjawiska rozwoju biologicznego człowieka. In: A. Malinowski and J. Strzałko (Eds.) *Antropologia* 1985:436–459. Warszawa-Poznań: Państwowe Wydawnictwo Naukowe.

Cieślik J., Mrowicka B.: Struktura zmienności fenotypowej względnej masy ciała w fazie ontogenezy progresywnej – wyodrębniona metodą Cole'a. In: P. Bergman, K. Kaczanowski, H. Piechaczek (Eds.) *Otyłość – epidemią XXI wieku. Dziewiąte Warsztaty Antropologiczne Im. Profesora Janusza Charzewskiego* 2006:12–23. Warszawa: Akademia Wychowania Fizycznego w Warszawie.

Cole T.J.: The LMS method for constructing normalized growth standards. *Eur J Clin Nutr,* 1990; 44:45–60.

Cole T.J, Green P.J.: Smoothing reference centile curves: the LMS method and penalized likelihood. *Stat Med* 1992; 11:1305–1319.

Kaczmarek M.: Longitudinal study of adolescent growth and biological maturation. *Acta Med Auxol* 2001; 33(3):205–211.

Kaczmarek M.: Wpływ warunków życia na wzrastanie i rozwój człowieka, Seria Antropologia nr 20. 1995. Poznań: Wydawnictwo Naukowe UAM.

Krawczyński M., Krzyżaniak A., Walkowiak J.: Normy rozwojowe wysokości i masy ciała dzieci i młodzieży miasta Poznania w wieku od 3 do 18 lat. *Pediat Prak* 2000; 4:341–353.

Wolański N.: *Rozwój biologiczny człowieka. Podstawy auksologii, gerontologii i promocji zdrowia*. 2005. Warszawa: Wydawnictwo Naukowe PWN.

World Health Organization.: *WHO Child Growth Standards: Methods and development: Length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age*. 2006. Geneva: World Health Organization.

Lewitt A, Mądro E, Krupienicz A.: Podstawy teoretyczne i zastosowania analizy impedancji bioelektrycznej (BIA). *Endokrynologia, Otyłość i Zaburzenia Przemiany Materii* 2007; 3(4): 79–84.

Magdalena Durda

# Biological status of adolescents in relation to their lifestyle behaviours and family's socioeconomic status

**Abstract:** The main objective of the study was to evaluate social correlates of the adolescent biological status, such as living conditions determined by SES of the family, and adolescents' own personal lifestyle.
The studied sample consisted of 2248 boys and girls aged 13 to 18 years, randomly selected from all types of school in Wielkopolska province within the framework ADOPOLNOR project. Data were collected between February 2009 and September 2010. In purpose to assess the adolescent biological status, anthropometric measurements of body height, weight and waist circumference (WC) were taken. The standardized questionnaires were used to assess: a) frequency of illnesses experienced by adolescents, b) SES of the family, c) adolescent lifestyle behaviours.
Computations were run using Statistica 9.0 Software. Statistical analysis of data comprised of unidimensional and multidimensional methods.
Stronger link between SES of the family and personal lifestyle was observed in boys, than in girls. Body height in both boys and girls were positively correlated with urban areas, as well as girls' maturation. Adolescents living in urban areas were more likely to have ill-health, compared to their peers living in countryside. Variability of somatic traits in boys was strongly associated with socio-economic status of their families. In girls, SES of family and their personal lifestyle operated synergistically. Frequency of illnesses in boys was modified mostly by lifestyle. Girls health was affected by both SES status of the family and lifestyle.
The findings revealed, that for purposes of assess biological status of adolescents it is best to consider both SES and lifestyle factors together.

**Key words:** adolescents, socioeconomic status, lifestyle, health-risk behaviours, ill-health

## Introduction

The period of adolescence is one of the key stages of human ontogeny, as it determines the acquisition of a biological and psychosocial ability to procreate and extend the species. It is the time when physiological and somatic traits of an adult in-

dividual are ultimately formed. Adolescence also offers the "last chance" to repair developmental defects that have occurred during the foetal stage, infantry and childhood and to create a phenotype that is capable of countering the impacts of adverse environmental conditions which are detrimental to an individual's health status, work ability and cognitive functions. The transition from a child to an adult is genetically determined and evolutionarily conserved. It is, however, subject to modifications caused by the impact of a broadly understood culture which may either help or hinder the genes to reveal their potential. The differences in the development of somatic traits are therefore regarded as sensitive indicators of society's level of well-being. Listed among cultural modifiers of biological status are living conditions determined by specific SES and lifestyle, as most significant differentiators of health status in particular stages of life cycle [Eiben and Mascie-Taylor 2004]. This means that biological and cultural aspects coexist and depend on each other. For that reason, human development, or actually human biology as a whole, should be considered from the biocultural perspective [Lasker 1969]. The impact of socio-economic environment on the variance of somatic traits is well known. The impact of the above-listed factors on the growth level is such that the higher the status of a social class, the bigger mean body height and weight values of its representatives [Goldstein 1971, Kaczmarek 1995]. This study puts a particular emphasis on the effect of the urbanisation factor on variation of biological traits. There is a lot of evidence indicating that rural areas in Poland are "impaired" in comparison to the city in respect of particular traits [Bielicki and Waliszko 1991]. While the effect of SES on the variance of biological traits in the period of adolescence is generally understood, lifestyle and its modifying role is yet to be evaluated. Understanding the relationship between adolescents' lifestyle and biological status seems to be all the more important considering that some spectacular processes occur at that time in young people's brains predisposing them to engage in health-risk behaviours. The changes relate to uneven development of the prefrontal cortex and the subcortical structures (part of the limbic system involved in the regulation of emotional behaviours and some emotional states, such as satisfaction, pleasure or fear) [Casey et al. 2008]. The adolescents' tendency to engage in health-risk behaviours more than in children and adults is accounted for by a relatively faster functional development of the affective and reward systems in the limbic system, as compared to the prefrontal cortex, resulting in a predisposition to take risky decisions and choices much more often than in children (in whom none of the listed elements is fully developed yet) and adults (whose fully developed limbic systems are sensitive to the activity of the reward system but able to control their drives owing to a mature prefrontal cortex) [Casey et al. 2008, Cohen et al. 2010]. And so health-risk behaviours may be deemed to arise from biologically generated drive to search for novelty combined with underdeveloped self-control skills. This inclination of adolescents seems to be particularly dangerous as it is likely to be consolidated and continued in further stages of life, increasing the risk of many morbidities, particularly those we have come to refer to as lifestyle diseases. Although adolescence is looked upon as a period of a relatively low morbidity and mortality, as compared to other age groups, many diseases, includ-

ing chronic ones, are rooted in that very period. Therefore, adolescence, which begins along with puberty, is essential for a long-term health of individuals [Buck and Ryan-Wenger 2003]. A number of lifestyle-related behaviours, such as smoking, premature initiation of sexual activity, low physical activity, and drinking alcohol, lead to the deterioration of health status both currently and in further stages of life. Health-risk behaviour is a characteristic manifestation of adolescence [Spear 2000], related to the performance of developmental tasks of that period, i.e.: searching for one's identity, autonomy, testing one's capabilities and stress management methods, performing new social roles [Jessor et al. 1987]. Engaging in health-risk behaviours, raising conflicts with parents and increased susceptibility to peer influence provoke young people to leave their families and start an independent life.

Results of physical development monitoring conducted under the National Health Programme are considered an important indicator of health status. In this context, studies on social and behavioural aspects of adolescent biological development are gaining particular relevance. The findings of this study allow to define the role of socio-economic factors and lifestyle in differentiating adolescents' biological status as manifested by particular somatic traits and the incidence of health problems. The significance of such studies appears even greater given that adolescence is the least explored of all ontogeny periods in this regard. Alongside with their cognitive importance, the results of this study form a sound basis for promoting disease prevention and healthy lifestyle among adolescents.

No research has so far been conducted to give a comprehensive picture of SES and lifestyle influence on the variability of biological traits in the period of adolescence. The problem seems all the more interesting as the difference of maturation rates of particular brain structures, reported by neurological studies; the development of self-identity, typical for this stage of life; increased influence of peers; and the tendency to part from one's family, all predispose adolescents to develop their own lifestyles, sometimes totally different than those pursued in their family homes. Therefore, this study attempts to investigate the relationships between the biological status of adolescent boys and girls, on one hand, and their lifestyles and families' socio-economic status, on the other. The main objective is to assess the association between the biological status of adolescent boys and girls and the lifestyle they pursue in a variety socio-economic conditions.

## Materials and methods

The investigations carried out under this study were part of the Polish-Norwegian research project called: At the doorstep to adulthood: adolescent health and quality of life in a variety of socio-economic backgrounds (ADOPOLNOR), PL0255. The sample consisted of 1231 boys and 1237 girls aged 13–18, students of lower and upper secondary schools. The participating adolescents came from Poznań, three former province capitals (administrative division before 1999): Kalisz, Konin and

Piła, and the villages of Biała, Bojanowo, Krajenka, Krzykosy, Lisków, Osieczna, Pięczkowo, Sulęcinek, Słupia Wielka. The study was cross-sectional in character. The study sample was based on a sampling frame developed for the purpose of the ADOPOLNOR project. Enrolled for the study were boys and girls whose parents had given a written consent for them to participate. Survey questionnaires were distributed among the selected students and their parents. Each questionnaire had completion instructions and basic information on the nature and objective of the study attached to it. Anthropometric examinations were performed by anthropologists and persons trained by them. Measurements were taken before noon, in school nursery rooms. The collection of data was started in February 2009 and completed in September 2010.

Somatic traits were measured according to a standard procedure [Martin and Saller, 1957], with upright stature and the head positioned in the Frankfurt plane, by measuring the height from the vertex point to the floor a subject was standing on. Body height was measured with a Swiss-made GPM anthropometer with the accuracy of 1 mm. Body weight was measured with medical scales (measurement accuracy of up to 100 g). Body height and weight values were used to establish BMI levels for each of the subjects. The BMI is set as an individual's body weight expressed in kilograms divided by the square of his or her height ($kg/m^2$). Due to a fast growth rate of children and adolescents, the BMI should be evaluated in correlation with age-specific centile distributions. Hence, children with underweight, overweight or obesity were identified with the Cole's method taking account of the the age-determined variation of body proportions. The structure of BMI centile distributions for both genders was made using the LMS method [Cole et al. 2000, 2007]. Each subject was measured for his or her Waist to Height Ratio, (WHtR). The WHtR is a simple marker distribution [McCarthy and Ashwell 2006]. The waist was measured by an elastic tape placed horizontally or somewhat obliquely midway between the lower edge of the ribs and and upper edge of the hip bone. The measurements were taken with subjects standing motionless, with the accuracy of 1 mm. The WHtR was set according to the following formula (I):

$$WHtR = WC/B\text{-}v \quad (I)$$

where: WC is waist circumference (in centimetres), B-v is body height (in centimetres).

A constant and uniform WHtR value was assumed for both genders (WHtR = 0.5), which amounts to the rule that abdominal obesity occurs with waist circumference (WC) exceeding the half of body height (B-v/2). Menarche age was determined based on the answers given by the participating girls, using the retrospective method.

In order to compare body heights of urban and rural boys and girls and in order to examine age-related changes in disease symptom incidence and lifestyle, the sample was classified into one-year age groups in such a way that particular age groups were middle parts of class intervals of boys and girls born during a given year, from January to December. Values of somatic traits were standardised to re-

move chronological age bias. Further analyses were then conducted on data converted to standardised residuals.

The boys' and girls' lifestyles, their families' socio-economic status and the incidence of health problems were investigated through survey questionnaires. Lifestyle was evaluated based on a standard Youth Quality of Life survey instrument (YQOL). The self-completion questionnaire contained a number of questions concerning lifestyle, with particular stress on health-related behaviour. The respondents were asked about a number of hours they spend every day in front of a computer/TV, physical activity (of at least 30 minutes during last seven days), smoking (including the age when the first cigarette was smoked), alcohol, drugs, sexual activity. The socio-economic status was determined based on the answers given by subjects' parents. The parents received a separate questionnaire where they responded to questions regarding place of residence, mother's and father's education, number of dependent children, number of working family members, number of bedrooms occupied by the family, and a subjective assessment of an income level. The questionnaire also served to examine the incidence of health problems. The occurrence of symptoms was indicated in a dichotomous scale, with 0 standing for the absence of a symptom and 1 meaning its presence. The questionnaire asked about the following symptoms: fatigue after exertion, weakness, frequent headaches, nose bleeding, pain in limbs and joints, stomach aches, vision disorders, paroxysmal sweating, urinary disorders, tachycardia, infections of upper respiratory tract and prolonged coughing.

The answers to these questions were provided by the parents.

The relevant database and all statistical analyses were made using the Statistica 9.0 software package. The normality of distributions was checked for continuous variables using the W Shapiro-Wilk test. The variance was evaluated with the Levene's test. Non-parametric tests: chi-square and Spearman's rank correlation were applied to determine the relationships between single categorised variables. A single-factor variance analysis was used to compare mean values in groups, for variables showing normal distribution. A double-factor analysis was conducted in order to demonstrate interaction between independent variables. A non-parametric equivalent of single-factor ANOVA, that is the Kruskal-Wallis rank test (H test) and median test, were applied for variables that met the criteria of normal distribution. Due to a comprehensive nature of the study and an aggregate nature of examined variables, a preliminary analysis was extended to comprise methods of multidimensional analysis, such as: correspondence analysis and multiple regression analysis.

## Results

Table 1 shows selected descriptive statistics for the sample.

In the first stage of the study, the normality of variable distributions was checked. The Shapiro-Wilk test showed that the distribution for body weight, BMI,

Table 1. Descriptive statistics for the sample

| Variables | Boys N=1231 | | Girls N=1237 | |
|---|---|---|---|---|
| | n | % | n | % |
| Age (in years) | | | | |
| 13.5 | 183 | 14.9 | 206 | 16.6 |
| 14.5 | 235 | 19.1 | 235 | 19.0 |
| 15.5 | 223 | 18.1 | 254 | 20.5 |
| 16.5 | 246 | 20.0 | 229 | 18.5 |
| 17.5 | 186 | 15.1 | 172 | 13.9 |
| 18.5 | 158 | 12.8 | 141 | 11.4 |
| BMI categories | | | | |
| Extreme underweight | 2 | 0.2 | 8 | 0.6 |
| High underweight | 14 | 1.1 | 21 | 1.7 |
| Underweight | 82 | 6.7 | 189 | 8.6 |
| Standard | 906 | 73.6 | 1845 | 75.9 |
| Overweight | 174 | 14.1 | 304 | 10.5 |
| Obesity | 45 | 3.7 | 75 | 2.4 |
| Place of residence | | | | |
| Urban area | 572 | 46.5 | 628 | 50.8 |
| Rural area | 659 | 53.5 | 609 | 49.2 |
| Smoking | | | | |
| No, never | 984 | 79.9 | 994 | 80.4 |
| I gave up smoking | 85 | 6.9 | 77 | 6.2 |
| I smoke | 156 | 12.7 | 161 | 13.0 |
| Drinking alcohol | | | | |
| No | 412 | 34.5 | 661 | 53.4 |
| Yes | 769 | 62.5 | 525 | 42.4 |
| No. of hrs. in front of TV/com. | | | | |
| 3 and less | 284 | 23.1 | 380 | 30.7 |
| 4–5 | 629 | 51.1 | 629 | 50.8 |
| 6 and more | 314 | 25.5 | 221 | 17.9 |
| Physical activity | | | | |
| High (4–7) | 694 | 56.4 | 456 | 36.9 |
| Medium (2–3) | 413 | 33.5 | 511 | 41.3 |
| Low (≤1) | 122 | 9.9 | 258 | 20.9 |
| Sexual activity | | | | |
| No, never | 994 | 82.4 | 1073 | 87.9 |
| Yes, a single time | 60 | 5.0 | 38 | 3.1 |
| Yes, more times | 152 | 12.6 | 109 | 8.9 |
| Taking drugs | | | | |
| No, never | 1086 | 92.0 | 1153 | 95.2 |
| Yes, a single time | 62 | 5.2 | 43 | 3.5 |
| Yes, more times | 33 | 2.8 | 15 | 1.2 |

WHtR and menarche age was not normal. The only variable to exhibit normal distribution was body height (W=0.999, p=0.539); therefore, it was only for that trait that parametric tests were used. For the other ones, the differences between urban and rural adolescents were examined with the non-parametric Kruskal-Wallis rank test and median test. Mean values and medians of those variables were calculated for all the age classes basing on current measurements of body height and weight. The variance analysis showed significant differences in body height between urban and rural boys and girls, applying to both genders in favour of urban adolescents. Rural boys were shorter than urban boys (F=10.2, p=0.001), and rural girls shorter than urban girls (F=9.0, p=0.003). A graphic representation of the two-factor analysis results are shown in Figures 1 and 2.

Rural and urban boys' and girls' body weight medians were compared using the median test, chi-square statistics and the Kruskal-Wallis rank test. The analyses showed no significant differences in both genders (boys: chi-square=1.59, df=1, p=0.207, H=2.3; p=0.122; girls: chi-square=0.02, df=1, p=0.887, H=0.03, p=0.858). However, the same statistical methods revealed a significant variation in the WHtR values between rural and urban girls. The urban ones were found to have lower WHtRs, thus showing lower accumulation of abdominal fat as compared to their rural peers (chi-square=3.31, df=1, p=0.068; H=4.45, p=0.035). No significant differences were found in WHtR values between rural and urban boys (chi-square=1.03, df=1, p=0.358; H=0.09, p=0.955). Another variable analysed was BMI. The chi-square test and Spearman's rank correlation showed signif-

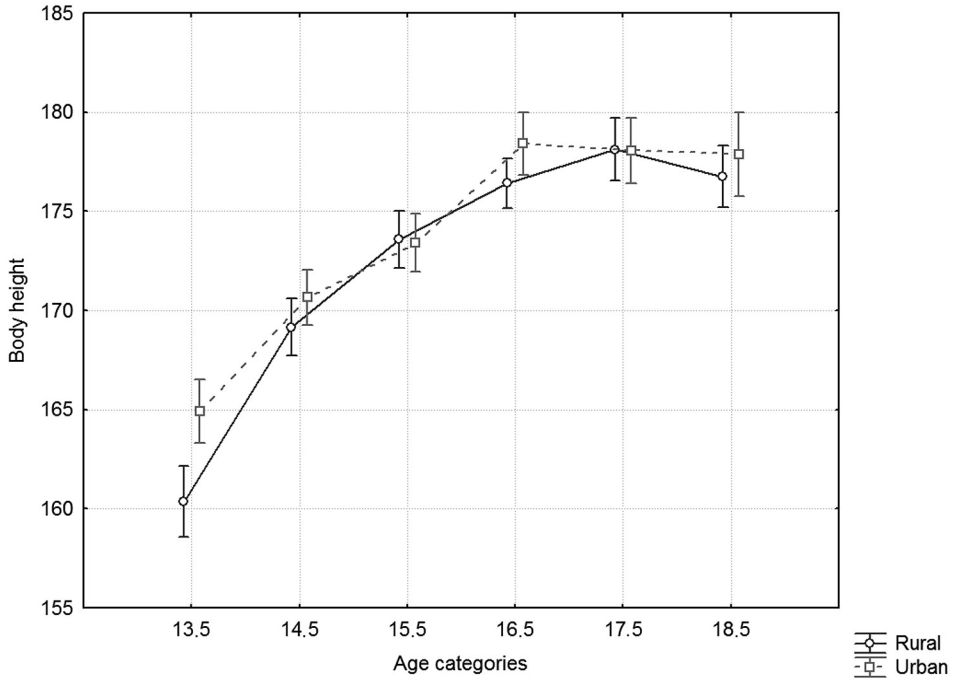



Fig. 1. Mean body height of urban and rural boys

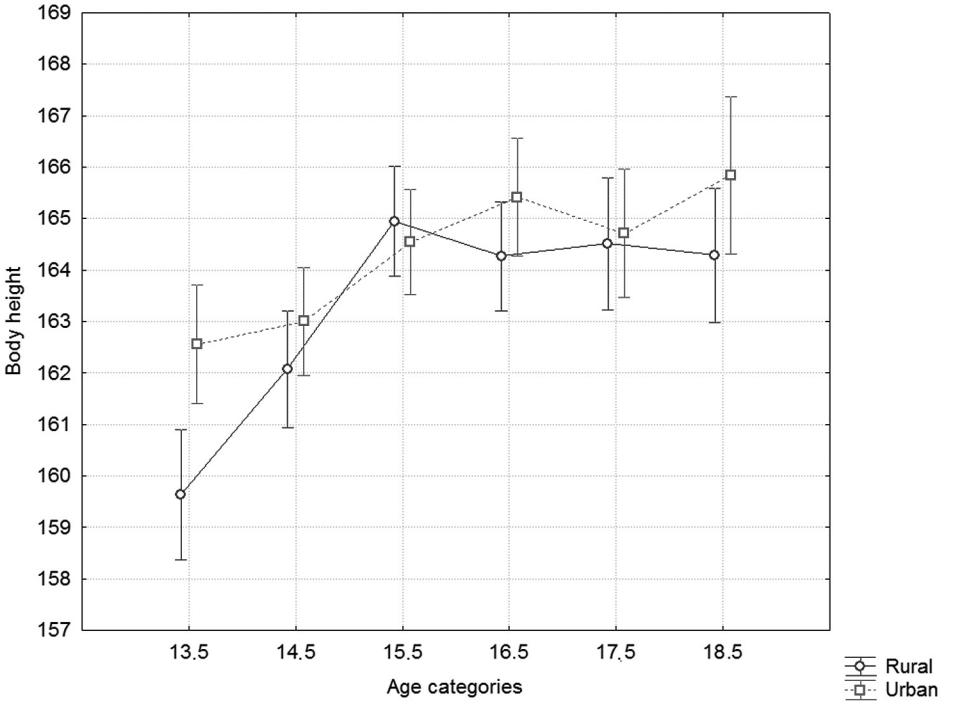



Fig. 2. Mean body height of urban and rural girls

icant differences between genders. Boys were characterised with higher BMIs compared to girls (chi-square=15.89, p<0.001; R=–0.08, p<0.001) and higher incidence of overweight (p=0.005). The analysis showed no significant differences in BMI between urban and rural adolescents. The only significant disparity being that rural boys were more likely to achieve the standard value of this indicator (p=0.018).

Menarche age served also as a positive indicator of a biological status. The average age of the first menstruation was 12.67 years (N=1086, Min=10.0, Max=16.5, SD=1.04), median 13.0 (Q1=12.0, Q3=13.2). The Kruskal-Wallis rank test showed that the place of residence significantly affects the age of the first menstruation (H=10.12, p=0.001). Menarche occurred earlier in girls living in the urban areas (Fig. 3)

Twelve self-reported symptoms of systemic disorders were analysed for the purpose of this study. Among the most prevalent symptoms were upper respiratory tract infections (41.8%), stomach ache (29.8%) and frequent headaches (27.8%). The least frequently reported were urinary disorders (1.7%). There were some differences in the prevalence of the symptoms between boys and girls. The girls were much more often affected with fatigue after exertion (chi-square=37.51; p<0.001), suffered from weakness (chi-square=47.53, p<0.001), had headaches (chi-square=34.64, p<0.001) and stomach aches (chi-square=137.5, p<0.001). Girls were also more often found to have nose bleeding (chi-square=6.14,

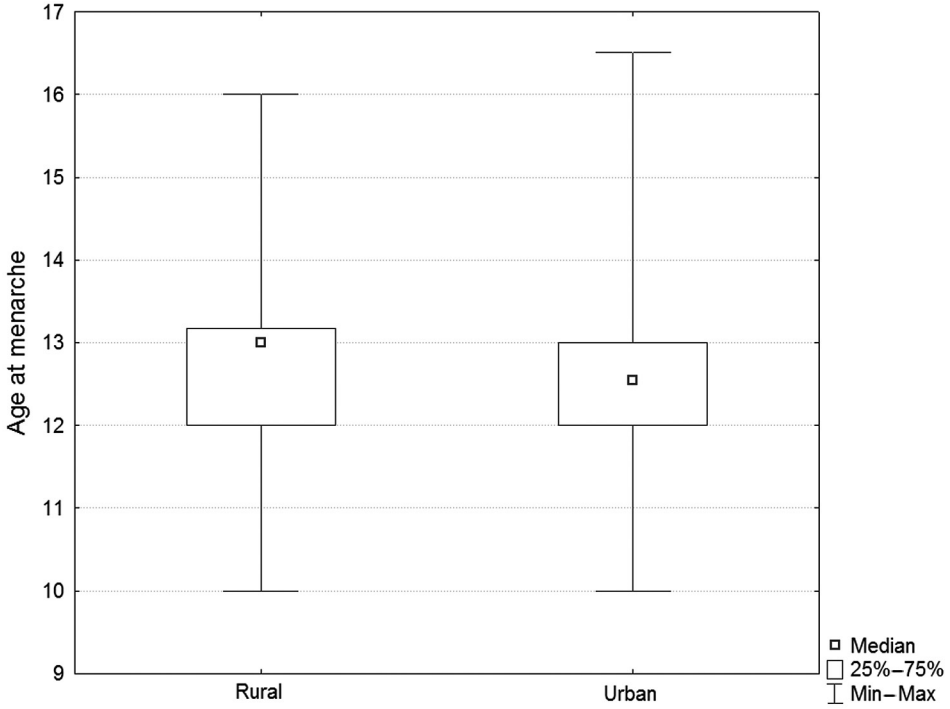



Fig. 3. Age at menarche in urban and rural girls

p=0.013), vision disorders (chi-square=8.02, p=0.005), urinary disorders (chi-square=8.04, p=0.005), and tachycardia (chi-square=6.92, p=0.008). In the course of further analysis, the difference in the incidence of systemic disorders in relation to place of residence was examined. Urban boys were found to suffer from upper respiratory tract infections (chi-square=29,31, p<0.001), stomach aches (chi-square=7.04, p=0.008) and weakness (chi-square=3.94, p=0.047) more often than rural boys. Mapped near the border of significance (p=0.055), chronic coughing affected urban boys more than their rural counterparts. Urban girls, as compared to their rural peers, showed a significantly higher incidence of three symptoms: limb and joint pain (chi-square=12.57, p<0.001), stomach ache (chi-square=4.49, p=0.034), and upper respiratory tract infections (chi-square=7.52, p=0.006). The analysis showed that urban population is characterised by a worse health status than rural population (see Fig. 4 and 5).

According to the information provided by the parents, the most common family model in the sample was that of two parents and two children. It accounted for 44.1% of all the families involved. Families with three children represented 40.0% and those with one child 13.0% of the total. 40.4% of the mothers had vocational or primary education, 36.8% secondary education, and 18.7% university education. A majority of the fathers had vocational/primary education (50.8 %). Those with secondary and higher education accounted for 31.85% and 11.0%, respectively. Most typically (44.9%), the study households had one income earner per

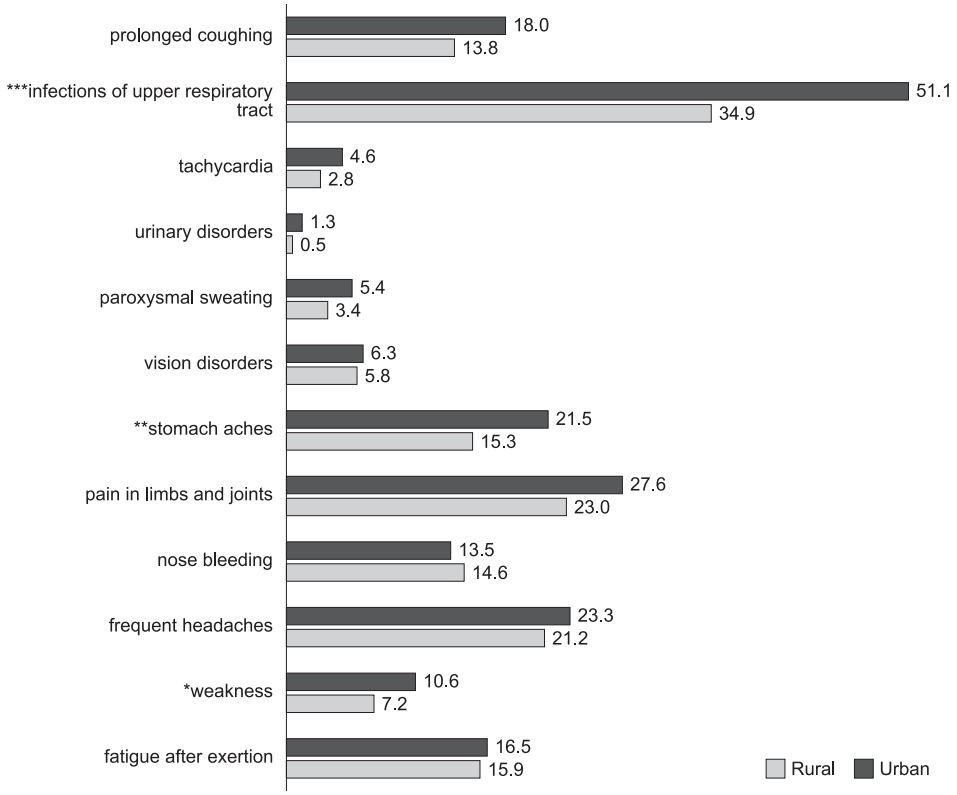



Fig. 4. Prevalence of diseases in urban and rural boys (*p<0.05; **p<0.01; ***p<0.001)

2–2.9 other persons. In 30.5% of the households, one working person supported 3 or more family members. Only 16.6% had one income earner per less than 2 family members. In terms of density, 37.8% of the households under study had less than 0.67 bedroom per person, 32.2% had 0.67 to 1 room per person, and 21.2% had more than 1 bedroom per person. A subjective measure of income sufficiency was used. Most of the parents reported to have sufficient resources to meet their needs fully (45.3%) or to a medium degree (36.1%). Almost 11% of the respondents admitted their household income was enough to meet only small part of their needs. There was also a small proportion of extreme cases of households with income levels meeting their needs in excess (2.4%) or not meeting at all (2.4%). A further stage of the analysis was aimed to examine the relationship between the socio-economic status and place of residence. To that end, chi-square and Spearman's rank correlation tests were used. The results achieved suggest a correlation between place of residence and all variables of socio-economic status. The parents of urban subjects were characterised by a significantly higher level of education. Their income level was also significantly higher. The number of children per household was significantly lower in the city than in the country, which was not reflected, however, in household density figures (Table 2).

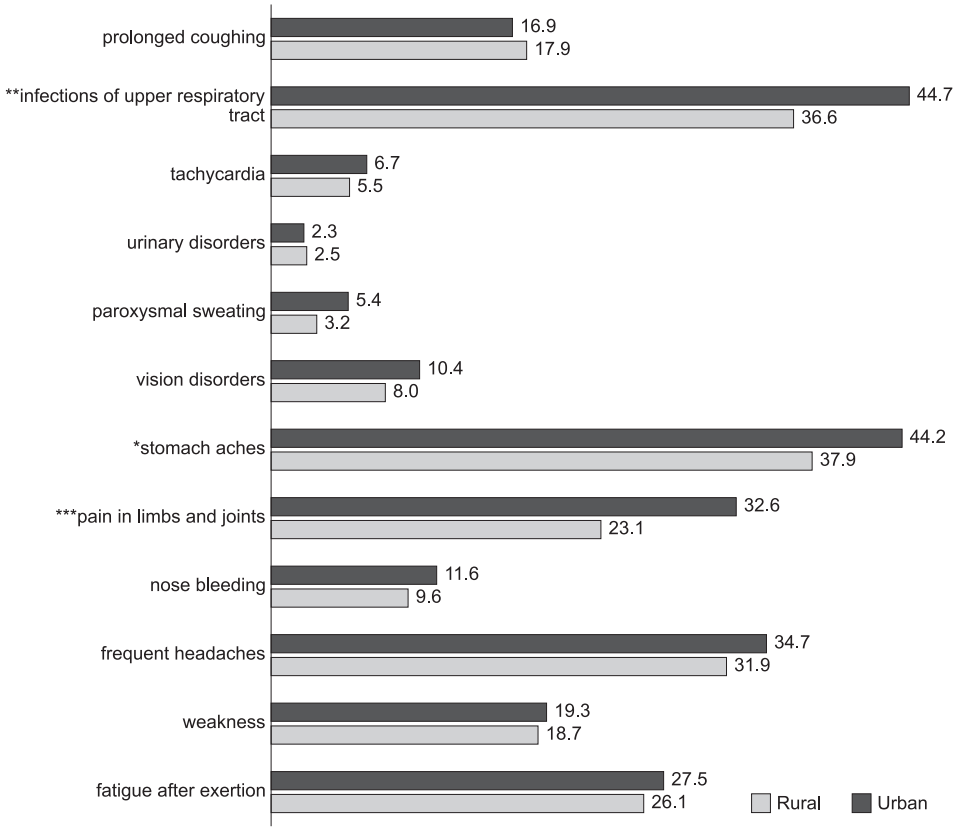



Fig. 5. Prevalence of diseases in urban and rural girls (*p<0.05; **p<0.01; ***p<0.001)

The study also analysed adolescent lifestyle-related variables, such as physical activity, daily number of hours spent in front of TV or computer, sexual activity, use of stimulants. The mean age for the first cigarette smoked was 13.2 years (Min=10, Max=8, SD=2.16). Girls were found to start smoking later than boys (boys: 12.99 years; girls: 13.45, F=9.11; p=0.003). More than a half of the subjects (52.4%) admitted drinking alcohol; 43.5% denied to have ever drunk alcohol. The analysis shows that boys use alcohol and drugs more often than girls (p=0.001 and

Table 2. Socio-economic variation of urban and rural families

| SES elements | chi2 | df | p | R | p |
|---|---|---|---|---|---|
| Mother's education | 158.43 | 2 | <0.001 | –0.26 | <0.001 |
| Father's education | 177.42 | 2 | <0.001 | –0.27 | <0.001 |
| Income | 6.81 | 1 | 0.009 | –0.05 | 0.009 |
| No. of children | 258.88 | 2 | <0.001 | –0.33 | <0.001 |
| No. of income earners | 68.74 | 2 | <0.001 | –0.17 | <0.001 |
| Household density | 39.78 | 2 | <0.001 | 0.12 | <0.001 |

p<0.001, respectively). They are also more active sexually (p<0.001) and spend more time in front of a computer or TV (p<0.001). Yet despite that tendency, they take intense physical exercise more than girls (p<0.001). No significant differences were found in the prevalence of smoking between boys and girls.

Another step was to examine the relationship between the adolescent lifestyle and place of residence. Again, chi-square and Spearman's rank correlation tests were used for that purpose. The results point out a significant correlation between place of residence and selected lifestyle variables. The group of boys displayed differences in the number of hours spent in front of TV/computer, use of drugs and sexual activity. Urban boys were found to spend significantly more time in front of TV/computer (p<0.001), more often reported to have taken drugs (p=0.013) and were less active sexually (p<0.001), as compared to rural boys. With girls, the differences showed primarily in the extent of drug use, the length of time spent in front of TV/computer, and smoking. Urban girls more often reported to have taken intoxicants (p<0.010), led more sedentary lifestyles (p<0.001) and smoked much more (p<0.001) than their rural peers (Table 3).

The mean age of sexual debut was found to be 16.1 years for girls and 15.4 years for boys. The difference in the age of sexual initiation between the boys and girls was statistically significant (F=15.2, p<0.001). The F test revealed a difference in the age of sexual initiation between urban and rural girls (F=11.42, p<0.001), with urban girls starting their sexual activity earlier (average values of girls' sexual initiation ages are 15.6 and 16.4 years, respectively). The age of sexual debut did not vary significantly between urban and rural boys (average values of boys' sexual initiation ages being 15.45 and 15.39 years, respectively).

One of the study questions regarded the relationship between a family's lifestyle and SES. To achieve an overview of the received results, a multidimensional

Table 3. Lifestyle variation of urban and rural adolescents

| Variables | $chi^2$ | df | p | R | p |
|---|---|---|---|---|---|
| | | | Boys | | |
| No. of hrs. in front of TV/com. | 25.93 | 2 | <0.001 | 0.14 | <0.001 |
| Taking drugs | 6.18 | 1 | 0.013 | 0.07 | 0.013 |
| Drinking alcohol | 1.06 | 1 | 0.302 | –0.03 | 0.303 |
| Sexual intercourse | 10.75 | 1 | 0.001 | –0.09 | 0.001 |
| Smoking | 2.87 | 2 | 0.238 | –0.01 | 0.604 |
| Physical activity | 2.33 | 2 | 0.312 | 0.04 | 0.204 |
| | | | Girls | | |
| No. of hrs. in front of TV/com. | 26.17 | 2 | <0.001 | 0.12 | <0.001 |
| Taking drugs | 6.53 | 1 | 0.010 | 0.07 | 0.010 |
| Drinking alcohol | 0.43 | 1 | 0.510 | 0.02 | 0.511 |
| Sexual intercourse | 1.09 | 1 | 0.296 | 0.03 | 0.296 |
| Smoking | 12.89 | 2 | 0.001 | 0.10 | <0.001 |
| Physical activity | 0.51 | 2 | 0.774 | –0.02 | 0.533 |

correspondence analysis was performed based on Burt's matrix (for each gender separately). The analysis employed only those SES and lifestyle variables which showed correlations in single analyses. In the analysis concerning boys, total randomness of chi-square statistics was explained by 24 dimensions, two of these, which explained together 22.4% of the variations, were selected for the analysis. These two dimensions were best set to explain the following categories of variables: mother's low education (quality=0.42), mother's high education (quality=0.40) and father's low education (quality=0.37). As far as girls are concerned, out of 24 dimensions explaining total randomness of chi-square statistics, two dimensions, represented by interrelated variables, were selected for further analysis. Those dimensions account for the largest proportion (26.0%) of the total variation. A quality analysis made for particular variables proved household density (quality=0.48), sexual activity (yes: quality=0.46, no: quality=0.46) and smoking (quality=0.44) to be best explained by both dimensions. Two separate groups of boys were identified by analysing the graphic representation of correspondence analysis results with regard to the first dimension. The first group comprised smokers (including those who had quitted smoking), sexually active, coming from families with few income earners, many children and high household density. Parents of the boys from that group had primary or vocational education. The other group included boys who did not smoke, were not sexually active and came from families with many budget contributors, few children, low household density and parents with secondary or high education. The interpretation of the graphic representation of the CA analysis for girls proved to be more difficult, as no clear-cut categories could be distinguished to show a strong relationship between lifestyle and socio-economic status variables. It could be observed, however, that the girls who engaged in health-risk behaviours, such as smoking, drinking alcohol, sexual activity, or drug use, were mapped far away from any other variables. (Fig. 6 and 7).

Therefore, it can be conservatively concluded that lifestyle factors in girls are less correlated with a socio-economic status than in boys.

Remarkably, lifestyle categories are aggregate in nature for both genders. Individuals who admit taking up one type of health-risk behaviour, usually tend to engage in other types, too.

A multiple forward stepwise regression analysis was applied to acquire a comprehensive view of the influence of the analysed SES and lifestyle variables on the menarche age and somatic trait variance in adolescents. By using the variance analysis, the goodness of fit was tested for the experimental data. The model eligibility criterion was set at F significance of $p<0.05$. None of the variables incorporated in the model showed collinearity. Since variables are known to interact with one another so as to strengthen or weaken their effect, the initial analysis (carried out separately for each gender) was fed with all the variables of socio-economic status (including the degree of urbanisation) and lifestyle (13 altogether). The linear fit between the models and the experimental data was good, as testified by variance analysis results (body height: boys $F=6.01$, $p<0.001$; girls $F=7.88$, $p<0.001$; body weight: boys $F=3.72$, $p<0.001$; girls $F=3.74$, $p<0.001$; BMI: boys $F=3.37$, $p=0.003$; girls $F=3.98$, $p<0.001$; WHtR: boys $F=2.64$, $p=0.033$; girls $F=5,38$,

p<0.001, menarche age: F=9.45, p<0.001). The models built for biological traits did little to explain the variation of those traits (for: body height 3% boys, 4% girls; body weight 3% boys, 4% girls; BMI 2% boys, 3% girls, WHtR 1% boys, 3% girls, menarche age 8%). Limited as they are in their explanatory role, the models do display correlations between SES and lifestyle, on one hand, and adolescent biological traits on the other hand. Boys' height was affected by the mother's education level (β=–0.11), girls' height, by the mother's education level (β=–0.10) and by physical activity (β=–0.11). Mother's higher education was a predictor of her children's higher body height. The same kind of correlation was found for physical activity: the higher physical activity of a girl, the taller she was likely to be. Boys' body weight was correlated with the mother's education (β=–0.08) and a number of children in the household (β=–0.09). For girls, it was correlated with the number

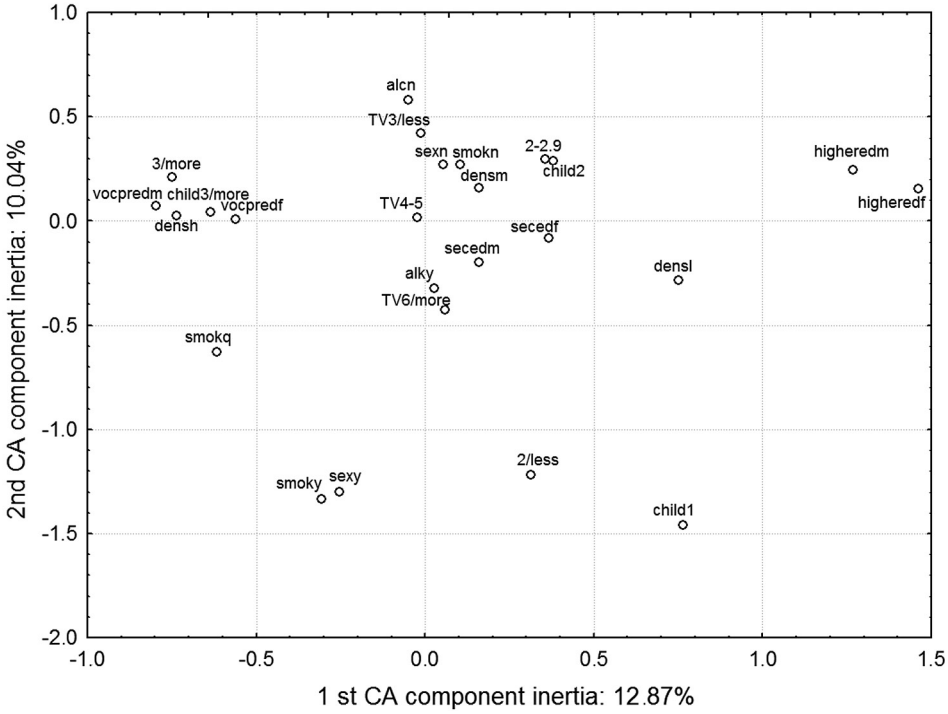



Fig. 6. Graphic representation of multidimensional correspondence analysis for lifestyle and SES variables for boys

higheredf – father's higher education, secedf – father's secondary education, vocpredf – father's vocational/primary education, higheredm – mother's higher education, secedm – mother's secondary education – mother's vocational/primary education, child1 – one child in the family, child2 – two children in the family, chld3/more – three and more children in the family, 3/more – ≥3 family members per income earner, 2–2.9–2–2.9 family members per income earner, 2/less – family members per income earner, densh – high household density (>1), densm – medium household density (2–0,7), densl – low household density (<0,07), alcn – does not drink alcohol, alcy – drinks alcohol, sexn – sexually inactive – sexually active, smokn – non-smoker, smokq – quitted smoking, smoky – smoker, TV3/less – no. of hours in front of a TV/computer 0–3/day, TV4–5 – no. of hours in front of a TV/computer 4–5/day, TV6/more – l no. of hours in front of a TV/computer 6 and more/day

of hours spend in front of TV/computer (β=0.09), father's education (β=0.10) and physical activity (β=–0.08). In boys, mother's higher education was associated with higher body weight. The relationship between the number of children in the family and boys' body weight was such that body weight decreased with the growing number of children in the family. Girls' body weight rose with father's decreasing level of education. Girls who spent much time in front of TV or computer tended to be heavier. Intense physical exercise was also found to be correlated with the growth of girls' body weight, which is likely to be due to the exercise-induced muscle growth. Boys' BMI, just as body weight, tended to be lower with individuals brought up in multi-children families (β=–0.08). Girls with low-educated fathers had higher BMIs (β=0.11), as did those pursuing a sedentary lifestyle (β=0.08). The girls who spent many hours in front of TV/computer were marked with higher BMI values.

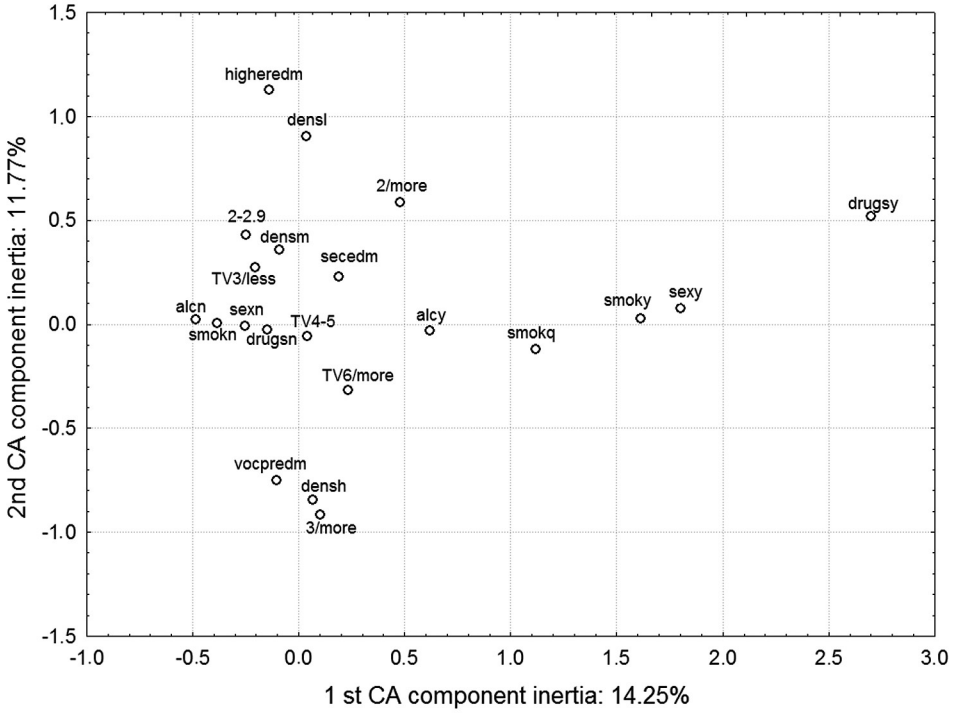



Fig. 7. Graphic representation of multidimensional correspondence analysis for lifestyle and SES variables for girls

higheredm – mother's high education, secedm – mother's secondary education, vocpredm– mother's vocational/primary education 3/more – >3 family members per income earner, 2–2.9–2–2.9 family members per income earner, 2/less – per income earner, densh – high household density (>1), densm – medium household density (2–0,7), densl – low household density (<0,07), drugsn – does not take drugs, drugsy – takes drugs, alcn – does not drink alcohol, alcy – drinks alcohol, sexn – sexually inactive, sexy – sexually active, smokn - non-smoker, smokq – quitted smoking, smoky – smoker, TV3/less – no. of hours in front of a TV/computer 0–3/day, TV4–5 – no. of hours in front of a TV/computer 4–5/day, TV6/more – no. of hours in front of a TV/computer 6 and more/day

A multiple regression analysis indicated a significant impact of SES and lifestyle variables on the WHtR in girls. Girls whose fathers had lower education were characterised with higher WHtRs (β=0.10), that is more abdominal obesity, than girls with higher educated fathers. The value of the ratio was also correlated with the number of hours spent in front of a TV/computer (β=0.07). Girls who led a sedentary lifestyle showed higher WHtRs. Boys exhibited no correlation in this respect.

The variation of menarche age were affected by the following variables: BMI, WHtR, urbanisation level, number of children in the family, and smoking. The age of the first menstruation negatively correlated with the BMI (β=–0.45) and urbanisation level (β=–0.09). Girls with higher BMIs had their first menstruation at a younger age. Girls living in the urban environment were also predisposed to a younger menarche age. Larger abdominal obesity was in turn associated with a later occurrence of the first menstruation (β=0.17). The girls brought up in multi-children families began their menarche age later (β=–0.07), as did those who smoked (β=–0.07).

The summary of the multiple regression analysis results for body height, body weight, BMI, WhtR and menarche age is shown in Table 4.

The same method of multiple regression was used to examine the impact of the lifestyle and SES variables on the incidence of health problems. The numbers of experienced symptoms were summed up for each subject to receive a set of scores ranging from 0 to 12. The aggregate score of symptoms constituted a dependent variable. Initially, the analysis (made separately for each gender) comprised all the variables of socio-economic status (including the level of urbanisation) and lifestyle, as well as biological variables, such as age, body weight and height, BMI and WHtR (18 variables altogether). The final model contained ten variables in boys and nine in girls. The variance analysis confirmed the goodness of fit to the experimental data (boys: $F=3.96$, $p<0.001$; girls: $F=5.59$, $p<0.001$). The model for boys

Table 4. Strongest modifiers of studied biological traits. Results of multiple regression analysed

| Variables | Body height | | Body weight | | BMI | | WHtR | | Menarche age |
|---|---|---|---|---|---|---|---|---|---|
| | boys | girls | boys | girls | boys | girls | boys | girls | |
| Mother's education | *** | ** | * | | | | | | |
| No. of children | | | * | | * | | | | * |
| Father's education | | | | ** | | *** | | * | |
| Income | | | | | | | | | |
| No. of hrs. in front of TV/com. | | | | ** | | * | | * | |
| Physical activity | | *** | | * | | | | * | |
| Urbanisation | | | | | | | | | * |
| Smoking | | | | | | | | | * |
| BMI | | | | | | | | | *** |
| WHtR | | | | | | | | | ** |

(*p<0.05, **p<0.01, ***p<0.001)

explained 5% of variances and the one for girls, 6%. A multiple regression analysis revealed that the incidence of systemic disorders in boys is affected by the level of urbanisation (β=0.13), smoking (β=0.10) and physical activity (β=0.07). In girls, the incidence of systemic disorders was correlated with five variables: physical activity (β=0.13), level of urbanisation (β=0.10), age (β=0.09), number of children in the family (β=–0.10) and number of employed persons in the family (β=0.08). A larger number of disease symptoms was associated with low physical activity, urban living environment and a small number of income earning members of the family. The age correlated with the quality of life in such a way that the older the girls were the more symptoms they reported. It was also found that girls from multi-children families suffered less from persisting pain and infections. Notably, two of the independent variables were found to be on the border of significance: BMI and father's education (health-related quality of life deteriorated with growing BMI and low level of father's education).

Table 5. SES and lifestyle variables that act as strongest modifiers of the incidence of reported symptoms. Results of multiple regression analysed

| Boys | Girls |
|---|---|
| urbanisation*** | physical activity*** |
| smoking** | urbanisation*** |
| physical activity* | age* |
| | no. of children* |
| | no. of income earners* |

(*p<0.05, **p<0.01, ***p<0.001)

Concluding, the incidence of systemic disorders in boys was to a larger extent associated with their lifestyles. The incidence of disease symptoms in girls was correlated with both lifestyle and their families' socio-economic status. Place of residence was a significant differentiator of disease incidence (Table 5).

## Discussion

The objective of this study was to assess the relationship between the biological status of adolescent boys and girls and the lifestyle they pursue in a variety of socio-economic conditions.

The study sample exhibited a gross disparity in adolescent behaviour depending on place of residence. Urban environment proved to be more conducive to health-risk behaviours for both boys and girls. Researchers have a mixed view of how adolescents' lifestyle is affected by their place of residence. Some authors endorse the results achieved in this study [Kishore et al. 1999]. Others, however, report opposite findings, claiming that rural youths are more likely to engage in health-risk behaviours or that place of residence does not matter for the incidence of health-risk behaviours [Levine et al. 2003]. Results of the above-mentioned authors have been received in studies conducted in various countries, with varied extent of differences in access to goods between urban and rural areas. Furthermore, in some reports, areas located on the outskirts of large cities were qualified as rural. It needs to be noted at this point, that the environmental importance of the ur-

banisation factor is strictly connected with a specific socio-economic system of a country. Countryside in Western Europe has a different social and economic dimension than that in developing countries. An important observation of this study is that boys and girls begin their sexual activity quite early. The mean age of sexual initiation in girls was 16.1 years and in boys 15.4 years. The age of the first sex has decreased substantially in the past several decades. Studies conducted by Durda et al. [2010] revealed the mean age of sexual debut for females born in the 1970s to be 19.1 years, while the mean sexual debut for females born in the 1990s is 16.3 years. The lowering age of sexual activity is a very alarming phenomenon. It is commonly believed that a low age of sexual initiation is one of the risk factors for unintended pregnancies in adolescents and sexually transferable diseases [Kirby 2002].

There were also significant differences in the frequency of health-risk behaviours between boys and girls. Boys were found to be more likely to engage in a risky lifestyle than girls. Studies conducted by Giedd et al. [1996] indicate that changes occurring in adolescent brains are more intense in boys, making them more predisposed to take risk and look for new experience. One might also venture to argue that there is more social acceptance for boys taking risky behaviours.

The multidimensional analysis examining the relationship between lifestyle and SES helped to reveal a system of interconnections between the two types of variables, thus showing differences between genders. Boys were more inclined to health-risk behaviours (smoking and sexual activity) if coming from families of a low SES. Girls, on the other hand, were found to engage in such behaviours regardless of their families' SES. Many reports have demonstrated that girls are more vulnerable to the pressure of mass culture promoting an attainable ideal of beauty which translates into a high degree of dissatisfaction with their bodies [Kaczmarek et al. 2008; McCabe and Riciardelli 2001]. Body self-image is the most important component of self-evaluation at puberty, and reduced satisfaction with one's appearance is a factor contributing to higher prevalence of health-risk behaviours. Rapid changes of body shape occurring during adolescence often lead to the body dissatisfaction, especially that young people show a strong tendency to compare themselves with both their peers and models presented by the media [Lokken et al. 2003]. A wrong self-image may lead to adverse emotional reactions (dissatisfaction, self-disappointment, depression). These may in turn reduce motivation to pursue health-promoting behaviours, such as following balanced diet or sustaining physical activity, and increased risk of engaging in health-risk behaviours (smoking, overusing alcohol, taking drugs). The literature of the subject also mentions increased aggressiveness of female adolescents in recent years which mainly accounted for by the modelling influence of the media that can also affect girls' lifestyles [Snethen and Puymbroeck 2008]. It needs to be added that girls tend to be more social-oriented than boys. They are known to attach more importance to peer relationships, which makes them more prone to adopting new lifestyles, often different than those practiced at home. The literature provides contradictory data on the relationship between adolescent lifestyle and socio-economic status of their families, whereas these two groups of factors are known to be closely related to each other in adults [Kaczmarek et al. 2006]. But adolescence is a special stage of

ontogeny, when young people develop their own patterns of behaviour under the influence of natural biological conditions and increasingly important peer groups, often discarding behaviour patterns they know from their family homes. Nevertheless, some authors argue that, despite a considerable impact of peer relations, there are still some associations between adolescent lifestyle and the family's socio-economic status [Geckova et al. 2002]. The wide-scale Health Behavior in School-aged Children (HBSC) Survey conducted in Poland by Mazur et al. [2007] revealed a correlation between health-risk behaviours and self-reported family SES. No relationship was showed, however, between the prevalence of those behaviours and objective indicators of a socio-economic status. Yet the study sample in that case consisted of 15-year-olds only. Some authors maintain that adolescents are so strongly affected by their peers that no other factors contribute to the development of their lifestyle, irrespective of their families' SES [Glendinning et al. 1995]. This difference of views probably arises from different methodological approaches (e.g. different ways of assessing family socio-economic status) hindering reliable comparison of results.

The analyses clearly indicated the persisting disparity in living conditions between the urban and rural population. In this regard, they confirmed results of other authors, including Olszewska and Łaska-Mierzejewska [2008], who have stressed that socio-economic discrepancy between the city and the country in Poland is much more pronounced than in highly developed countries of the European Union. The difference in socio-economic status between the two living environments was reflected in the values of biological traits: body height, menarche age and WHtR. This proves that social gradients based on urbanisation levels are still observed in Poland. They have been reported in a number of studies, including Hulanicka et al. [1990], Bielicki and Waliszko [1991], Bielicki et al. [1997], Bielicki and Szklarska [1999], Olszewska and Łaska-Mierzejewska [2008]. The authors indicate larger body size, particularly linear, including body height, in urban children and adolescents. They also show a tendency in urban girls to enter puberty earlier. In our study, the mean menarche age in urban girls was 12.58 (Me=12.54) years versus 12.76 (Me=13.00) years in rural girls. The age of first menstruation in urban girls was unusually low. As shown by the study carried out by Kaczmarek [2001], the mean age of the first menstrual bleeding for girls living in Poznań was 12.87 years. The fall of menarche age is most commonly explained by improvement of living conditions and has been documented in the literature of the subject [Łaska-Mierzejewska 1983, Bielicki et al. 1986, Cichocka and Żarów 2002]. According to Bielicki and Szklarska [1999], earlier occurrence of menarche age in urban girls results, among other factors, from a better access to health care and better sanitary conditions than in rural areas. On the other hand, studies by Cichocka and Żarów [2002] reveal that early sexual maturation is associated with the factors which are known to be harmful to human physical and mental health. Urban girls have shown a lower degree of abdominal obesity, which means they are slimmer than their rural peers. This trend (slimming figure) has also been noted by other authors [Palczewska et al. 2000, Eiben and Mascie-Taylor 2004] and may be due to the fact that adolescents are put under increasing pressure to have slim bodies.

This for the most part applies to girls [Kaczmarek et al. 2008], particularly those living in the city. The study demonstrated no differences in body weight and BMI, although in some calculations those parameters oscillated around the significance border. The fact has also been confirmed by other authors [Kumar et al. 2004, Oblacińska and Jodkowska 2007], proving the abatement of differences in selected biological and traits between the urban and rural living environments.

In terms of self-reported health problems, a strong discrepancy was revealed between the city and the country, but this time in favour of the latter. Significant differences were found in the incidence of respiratory tract infections, weakness, stomach ache and limb and joint pains. There was, however, a tendency for an increased incidence of all the studied disorders in urban areas, the differences not being significant though. It may be assumed that the city is a more stressful environment to live in. Subjective as they were, self-reported data on the incidence of systemic disorders collected for the purpose of this study confirm the variation in health status between Polish urban and rural children and adolescents. According to the report on Polish population's health status, carried out in 2004 by the Central Statistical Office, the city as a living environment contributes to the increased risk of chronic diseases. A higher incidence of health problems in urban areas is due to a more elevated level of pollution and noise as compared to a rural setting. In the province of Wielkopolska, the emission distribution corresponds to the extent of urbanisation. The highest emission from point sources and a heavy linear emission are found in urbanised areas of the central Wielkopolska, large cities and the industrial zone in the eastern part of the province [www.gios.gov.pl]. Noise seems to be another important source of stress. Noise measurements and surveys made in Poznań over past few years indicate that the city has an unfavourable acoustic climate. This applies mostly to the centre and the districts located along main roads. As reported by acoustics research, the main transit thoroughfares, roads leading to the centre and some inner streets generate noise exceeding accepted daytime limits (75–80 dB versus the functionality-dependent limit of 50–65 dB) [Environmental Protection Programme for the City of Poznań, www.poznan.pl]. Urban environment poses other threats, too. In the study made by Mazur [2007], young city dwellers complained more often about unsatisfactory relations with their parents, and consequently suffered more from the lack of emotional security, ever so important in that stage of life. Assuming that an ultimate manifestation of an organisms' adjustment to changing environmental conditions is the efficacy of their functioning, rather than just body size [Krawczyński 2003], a question arises of whether the urban environment, despite its abundance of goods and better access to health care and education, is rightly referred to as "better"? And going further: is it right to judge the quality of a living environment only by somatic traits achieved by its inhabitants? There a distinct discord between larger body size and poorer health status of urban adolescents. The rate of cultural change in the city may be so fast and the pressure so heavy that a human organism is not able to adapt to them, responding by reduced immunity manifested with symptoms like infections, weakness and pains. Variations in the rate of cultural and biological processes, although in a somewhat dif-

ferent aspect, has been discussed in the studies by Kościński [2001] and Garruto et al. [2004].

A comprehensive analysis of the role of socio-economic status and lifestyle factors in modifying the variance of adolescent biological status was the main objective of this study. Body height is believed to be a sensitive indicator of living conditions [Bielicki et al. 1986, Kaczmarek 1995]. However, the external environment modifies the degree in which genetic potential is released, and environmental factors may work with different intensity. Of all variables included in our analysis, mother's education proved to be the strongest modifier in boys, whereas physical activity and mother's education, in girls. These findings confirm that mother's education has a stronger impact on the development of children's linear body size than father's education. In the context of this variable, a gradient growth of body height is observed in both boys and girls. This comes in line with the observations of Bielicki et al. [1997], as well as Kozieł and Lipowicz [2009]. The impact of mother's education on the physical development status is accounted for by mother's better ability to improve the quality of diet and reduce morbidity rate in the childhood period [Frost et al. 2005].

A comprehensive analysis of the impact of cultural variables on girls' body height showed a highly significant relationship between this trait and physical activity. There have been a large number of reports concerning the effect of physical activity on girls' body height. Depending on the type and intensity of exercise, physical activity has been shown to have either inhibiting or stimulating effect. While girls practising competitive, high-performance sports (gymnasts) usually had lower body heights, those doing less demanding sports tended to have higher body heights [Malina 1994]. Reasonable and age-adjusted movement activity is a factor contributing positively to biological development, as it boosts the growth of the musculoskeletal system. The positive impact of movement activity on body height is explained by its stimulating effect on GH excretion [Malina et al. 2004]. Eliakim et al. [1996] published results showing that sporting girls showed elevated levels of GH, GH binding protein (GHBP) and IGF-1 in their blood. It is hard to explain why this factor proved to be significant for the variation of this trait in girls only. The multiple regression analysis revealed that father's education, number of hours spent in front of a TV/computer and physical activity were modifying factors for body weight in girls. Boys' body weight was proved to be affected only by their families' socio-economic status variables, i.e. mother's education and number of children in the family. The impact of mother's education on boys' body weight is explained by mother's ability to ensure appropriate diet, as in the case of body height. It is also well known that a size of the family significantly affects body weight and is a strong modifier of that trait. Mean body weight decreases with the growing number of children in the family [Eiben and Mascie-Taylor 2004]. The results of this study thus confirm the findings of other authors. The predominance of SES variables in the development of body weight variation in boys may be accounted for by a higher ecological sensitivity and lower developmental stability of adolescent males as compared to females. Many studies have demonstrated boys' being more sensitive to such environmental factors as nutrition or climate [Komlos and Lauderdale 2007].

A comprehensive analysis of the impact of SES and lifestyle variables indicated the number of children in the family as the most important modifying variable for the BMI in boys and the number of hours spent in front of a TV/computer and father's education, in girls.

The uncovered correlations between lifestyle variables and the BMI suggest that in this stage of ontogeny certain behaviours already begin to affect the current and future health status. In adult life, lifestyle is considered to be the most important health contributing factor [Lalonde 1987]. It is also agreed that higher body weight during adolescence is a risk factor for obesity and cardiovascular diseases in further stages of life [Wang et al. 2008]. The analysis of WHtR variability in girls showed that the index was associated with father's education, income level and number of hours in front of a TV/computer. In boys, none of the variables affected the variance of that index. Therefore, father's education may not be disregarded as an important modifier of particular somatic characteristics in girls. Father's education is thought to have an indirect effect by determining the way material goods are used, defining the level of health culture in the family and, increasingly so, by developing health-promoting attitudes in children [Siniarska 1994].

Discussing the results of the study, one has to note contradictory directions of the impact that parents' education exerts on female and male adolescents' body weight variation. On the one hand, mother's higher education predisposed boys to achieve heavier body weight. On the other hand, girls' body weight rose with their father's decreasing level of education. The results of this study are ascertained by the literature that reports obesity and overweight to often occur in social groups of extreme nature (high or low status) [Hakeem 2001]. The influence of father's education on female children's body weight was discussed, *inter alia*, by Kozieł and Lipowicz [2009]. According to Siniarska [1994], girls are more sensitive to their families' living conditions for which the father is mostly responsible.

In conclusion, it needs to be said that selected factors of socio-economic status and lifestyle modified adolescent physical development with varied intensity. The variance of somatic traits in boys was mostly affected by variables of their families' socio-economic status (mother's education and number of children in the family). Girls biological variables were modified to the greatest extent by lifestyle factors (number of hours spent in front of a TV/computer and physical activity) and father's education. The development of physical traits in girls is then determined by a synergic action of the two types of variables.

The study also investigated the impact of a variety of factors (biological, SES, lifestyle) on the onset of girl's puberty. Menarche age is used as an indicator of environmental conditions prevailing over the period of childhood and treated as very sensitive index of sexual development level [Cameron and Nadgdee 1996]. The age of the first menstruation is believed to depend on most sensitive indicators of socio-economic situation, and a low menarche age reflects well-being of society [Lindgren 1976]. The age of the first menstrual bleeding is conditioned genetically and environmentally. In this study it showed correlation with BMI, WHtR, urbanisation level, number of children in the family and smoking. There have been many reports proving the impact of obesity on the onset of girls' puberty. A high level of

body fat is known to trigger earlier puberty. This is most likely related to the reaction occurring in the peripheral fat tissue which involves aromatisation of adrenal androgens to oestrogens induced by aromatase [Zarzycki 1992]. Only one SES variable, namely the number of children in the family, was found to affect menarche age. A larger number of children in the family caused delay in menarche age, likely due to reduced quality and quantity of food per child. The more children in the family the poorer the diet. As a consequence, those girls achieve lower levels of body fat, which postpones the time of the first menstrual bleeding. The delay in menarche age in girls brought up in multi-children families was documented by Dann and Roberts [1984], and Cameron and Nadgdee [1996]. Results of this study indicated a later menarche age in girls with larger degree of abdominal obesity. The literature provides research results that affirm association between oestrogen level and a type of fat tissue distribution. It is believed that a high level of oestrogens a induces a female-type fat tissue distribution, that is deposition of fat in the lower part of the body. A central type of fat distribution is characteristic of females with lower levels of that hormone [Cucinelli et al. 2002]. A lower level of oestrogens in girls characterised by central fat distribution may predispose them to a later attainment of sexual maturity. The study revealed a relationship between smoking and menarche age. Smoking caused delay in the first menstrual bleeding. The literature reports on results regarding the exposure to tobacco smoke and its impact on the age of first menstruation. For all the difference of opinions, the authors of the most recent studies on that issue are inclined to admit that the exposure to tobacco smoke delays the first menstruation [Ferris et al. 2010]. Tobacco smoke contains over 400 chemicals, including polycyclic aromatic hydrocarbons (PAH) responsible for destruction of primary ovarian follicles, thus contributing the lowering of oestrogen level and earlier menopause [Shiveric and Salafia 1999]. Ferris et al. [2010] put forward a view that since a reduced level of oestrogens accelerates menopause, it may just as well have a delaying effect on menarche age.

Girls in this study reported symptoms of health issues much more often than boys, following the pattern described in other studies [Mazur 2007]. There have been many attempts to explain this phenomenon. Some researchers referred to genetic set-up, a different perception of changes occurring in the body and cultural environment. Most probably, however, all these factors add up to make girls more predisposed to systemic disorders than boys. The incidence of self-reported disease symptoms was affected by both SES and lifestyle variables. The impact of lifestyle was stronger than for earlier discussed variables. The differentiating factors in both genders included: urbanisation and physical activity. Both urban setting and low physical activity were predictors of a worse health status. In addition, boys' health was adversely affected by smoking, and girls' health by progressing age and a low number of income earners in the family. The number of children in the family represented an exception. A larger number of children in a family translates into lower incidence of symptoms, probably because the attention of parents in multiple-children families becomes more dispersed. A strong correlation between lifestyle factors and disease symptoms underscores an important health modifying role of those factors already in the stage of adolescence. Our results in-

dicate a synergic role of SES and lifestyle variables in contributing to negative indicators of biological status in girls. The literature provides contradictory data in this area. Some authors are of the view that there are some social gradients of adolescent health status [Torsheim et al. 2004]. Others claim that correlation either does not exist at all or is very weak [West 1997]. In their opinion, a growing independence from parents and larger share of one's own lifestyle in the development of health status bring about equalisation of social discrepancies. The strength of the correlation between socio-economic and health statuses depends to a large extent on country of study. Social predictors of health are not observed in highly developed countries (UK, Nordic countries). They are, however, found in poorer countries, which may be accounted for by strong social contrasts and consequent inequalities in the access to health care or education.

According to Bielicki et al. [1997], Polish population is highly homogeneous in genetic terms. Therefore, variances of polygenically determined traits are affected by modifying effects of environmental factors.

Family's SES is manifested through generally understood hygiene, behaviour patterns, diet. Lifestyle affects the body more directly (e.g. physical activity lowers high blood pressure, strengthens the muscles and stimulates the hormonal system). In view of the above, research into adolescent lifestyle seems to be of great importance. Behaviour patterns developed in the course of stormy transformations are often consolidated and continued over further stages of life. It needs to be borne in mind that adolescent males and females are biologically predisposed to engage in health-risk due to uneven development of particular brain structures. Reduced motivation, another side effect of adolescent neurobiological development, may additionally encourage young people to give up physical activity, particularly in view of unlimited access to a computer or TV, the two gods of sedentary lifestyle.

The joint contribution of lifestyle and socio-economic status variables to the development of adolescent biological status confirms the view that human biology should be considered in a biocultural perspective.

## Conclusion

1. The relationship between family socio-economic status and one's lifestyle is different in boys and girls. Boys' lifestyles are associated with their families' socio-economic status, a low SES being conducive to health-risk behaviours. Girls engage in such behaviour regardless of their families' socio-economic status.
2. Biological status of boys and girls shows different degrees of correlation with their families' socio-economic status and their own lifestyle. Boys tend to be more sensitive to living conditions resulting from their families' socio-economic status than to their own health-related behaviours. This emphasises a superior role of SES factors among other environmental modifiers of boys' physical development. Biological status of girls depends both on their families' socio-economic status and their own lifestyle.

3. The effect of the urbanisation factor on adolescent biological status is unclear. Urban adolescents are taller than their rural peers. Urban girls attain their sexual maturity earlier than rural ones. On the other hand, urban boys and girls are characterised by a worse health status as compared to their peers living in the country.

## References


Bielicki T., Waliszko H.: Urbanization-dependent gradients in stature among Polish conscripts in 1976 and 1986. *Am J Hum Biol* 1991; 3:419–424.

Bielicki T., Szklarska A., Welon Z., Brajczewski C., Nierówności społeczne w Polsce: antropologiczne badania poborowych w trzydziestoleciu 1965–1995. *Monografie Zakładu Antropologii PAN* 21, 1997. Wrocław.

Buck J.S., Ryan-Wenger N.A.: Early adolescents' definition of health: the development of a new taxonomy. *JTCT* 2003; September 22.

Cameron N., Nadgdee I.: Menarcheal age in two generations of South African Indians. *Ann Hum Biol* 1996; 23:133–119.

Casey B.J., Getz S., Galvan A.: The adolescent brain. *Dev Rev* 2008; 28:62–11.

Cichocka B., Żarów R.: Zmiany sekularne wieku menarche u dziewcząt z Krakowa, Warszawy i Wrocławia w latach 1965 – 2000 a sytuacja psychosocjalna. *Pediatr Pol* 2002; 4:317–322.

Cohen J.R., Asarnow R.F., Sabb F.W., Bilder R.M., Bookheimer S.Y., Knowlton B.J., Poldrack R., A unique adolescent response to reward prediction errors. *Nat Neurosci* 2010; doi: 10.1038/nn.2558.

Cole T.J, Flegal K.M., Nicholls D., Jackson A.A.: Body mass index cut offs to define thinness in children and adolescents: international survey. *BMJ* 2007; 335: 194, doi: 10.1136/bmj.39238.399444.55.

Cole T.J., Bellizzi M.C., Flegal K.M., Dietz W.H.: Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 2000; 320:1240–1243.

Cucinelli F., Soranna L., Barini A., Perri C., Leoni F., Manusco S., Lanzone A.: Estrogen treatment and body FAT distribution are involved in corticotropin and cortisol response to corticotropin-releasing hormone in postmenopausal woman. *Metabolis* 2002; 51(2):137–143.

Durda M., Jarząbek-Bielecka G., Kaczmarek M.: Patterns of sexual activity among young Polish women born between 1975 and 1995. Abstract, 17th Congress of the European Anthropological Association: Biological, Social and Cultural Dimensions of Human Health, 29.08–2.09.2010. Poznań UAM, Book of Abstracts:36.

Eiben O.G., Mascie – Taylor C.G.N.: Children's growth and socio – economic status in Hungary. *Econ Hum Biol* 2004; 2(2):295–320.

Eliakim A., Brasel J.A., Mohan S., Barstow T.J., Berman N., Cooper D.M.: Physical fitness, endurance training, and the growth hormone – insulin-like growth factor I system in adolescent females. *JCEM* 1996; 81:3986–3992.

Ferris J.S., Flom J.D., Tehranifar P.T., Mayne S.T.: Terry M.B.: Prenatal and childhood environmental tobacco smoke exposure and age at menarche. *Paediatr and Perinat Ep* 2010; 24:515–523.

Frost M.B., Forste R., Haas D.W.: Maternal education and child nutritional status in Bolivia: finding the links. *Soc Sci Med* 2005; 60:395–407.

Garruto R.M., Little M A., Weitz C.A.: Environmental stress and adaptational responses: Consequences for human health outcomes. *Coll Antropol* 2004; 28(2): 509–540.
Geckova A.M., vad Dijk, Groothoff J.W., Post D.: Socio-economic differences in health risk behaviour and attitudes towards health risk behaviour among Slovak adolescents. *Soz Praventiv Med* 2002; 47(4):233–239.
Giedd J.N., Snell J.W., Lange N., Rajapakse J.C., Casey B.J., Kozuch P.L., Vaituzis A.C., Vauss Y.C., Hamburger S.D., Kaysen D., Rapoport J.L.: Quantative magnetic resonance imaging of human brain development: ages 4–18. *Cereb Cortex* 1996; 6(4):551–560.
Glendinning A., Hendry L., Shucksmith J.: Lifestyle, health and social class in adolescence. *Soc Sci Med.*1995; 41(2):235–248.
Goldstein H.: Factors influencing the height of seven-year-old children – results from the National Child Development Study. *Hum Biol* 1971; 43(1):92–111.
Hakeem R.: Socio-economic differences in height and body mass index of children and adults living in urban areas of Karachi, Pakistan. *Europ J Clin Nutr* 2001; 55:400–406.
Jessor R.: Problem-behavior theory, psychosocial development, and adolescent problem drinking. *Br J Addic* 1987; 82:331–342.
Kaczmarek M.: *Wpływ warunków życia na wzrastania i rozwój człowieka*, Wydawnictwo Naukowe UAM, Poznań, 1995.
Kaczmarek M., Durda M., Krzyżaniak A.: Is gender a crucial cause of body image in adolescence? *Pol J Environ Stud* 2008; 17(4A):182–186.
Kaczmarek M., Skrzypczak M., Maćkowiak K.: Status społeczno – ekonomiczny oraz styl życia jako czynniki różnicujące subiektywne poczucie zdrowia wśród starzejących się mężczyzn. *Gerontol Pol* 2006; 14(2):84–90.
Kirby D.: Antecedents of adolescent initiation of sex, contraceptive use, and pregnancy. *Am J Health Behav* 2002; 26(6):473–485.
Kishore J., Sing A., Grewal I., Snigh S.R., Roy K.: Risk behaviour in an urban and a rural male adolescent population. *Natl Med J India* 1999; 12(3):107–110.
Komlos J., Lauderdale B.E.: Underperformance in affluence: the remarkable relative decline in American heights in the second half of the 20th– century. *Soc Sci Quart* 2007; 88(2):283–304.
Kościński K.: Is there a relationship between social adjustment and physique? *Var Evol* 2001; 9:5–18.
Kozieł S, Lipowicz A.: Concurrent effect of social factors and maturity status on height and BMI of adolescent girls. *J Life Sci* 2009; 1(2):133–137.
Krawczyński M.: Wzrastanie, dojrzewanie i sprawność fizyczna dzieci i młodzieży w Polsce na przełomie XX i XXI wieku. *Endokrynol Ped* 2003; 1(2):9–16.
Kumar B.N., Holmboe-Ottesen G., Lien N., Wandel M.: Ethnic differences in body mass index and associated factors of adolescents from minorities in Oslo, Norway: a cross national study. *Public Health Nutr* 2004; 7(8):999–1008.
Lalonde M.: *A new perspective on the health of Canadians*, Ministry of Supply and Services, Ottawa, 1974.
Lasker G.W.: Human biological adaptability. *Science* 1969; 166:1480–1486.
Levine S.B., Coupey S.M.: Adolescent substance use, sexual behavior and metropolitan status: Is "urban" a risk factor? *J Adol Health* 2003; 32:350–355.
Lindgren G.: Height, weight and menarche in Swedish urban school children in relation to socioeconomic and regional factors. *Ann Hum Biol* 1976; 3:305–528.
Lokken K, Ferrano F.R., Kirchner T., Bowling M.: Gender differences in body size dissatisfaction among individuals with low, medium, or high levels of body focus. *J Gen Psychol* 2003; 130(3):305–310.
Łaska-Mierzejewska T.: Wpływ społecznego zróżnicowania ludności wiejskiej na wiek menarche i jego trend sekularny, *Mat Prac Antropol* 1983; 103:21–43.

Malina R.M.: Physical growth and biological maturation of young athletes. *Exerc Sport Sci Rev* 1994; 22:389–443.
Malina R.M., Bouchard C., Bar-Or O.: *Growth, Maturation and Physical Activity*. 2004. Champaign: Human Kinetic.
Martin R., Saller K.: *Lehrbuch der Anthropologie*, Band I, Gustav Fisher Verlag, Stuttgart, 1957.
Mazur J.: *Status materialny rodziny i otoczenia a samopoczucie i styl życia młodzieży 15-letniej*. Instytut Matki i Dziecka, Warszawa, 2007.
McCabe M.P., Ricciardelli L.A.: Parent, peer and media influences on body image and strategies to both increase and decrease body size among adolescent boys and girls. *Adolescence* 2001; 36:225–240.
McCarthy H.D., Ashwell M.: A study of central fatness using waist-to-height ratios in UK children and adolescents over two decades supports the simple message – 'keep your waist circumference to less than half your height'. *Int J Obes (Lond)* 2006; 30(6):988–92.
Olszewska E., Łaska-Mierzejewska T.: Unemployment in the Polish countrywide and its effect on the development and rate of maturation of rural girls. *Anthropol Rev* 2008; 71:33–42.
Piko B., Fitzpatrick K.M.: Does class matter? SES and psychosocial health among Hungarian adolescent. *Soc Sci Med* 2001; 51:817–30.
Program ochrony środowiska dla miasta Poznania, available at:
Shiverick K.T., Salafia C.: Cigarette smoking and pregnancy I: ovarian, uterine and placental effects. *Placenta* 1999; 20:265–272.
Silberg J., Pickles A., Rutter M., Hewitt J., Simonoff E., Maes H., Carbonneau R., Murrelle L., Foley D., Evas L.; The influence of genetic factors and life stress on depression among adolescent girls *Arch Gen Psychiat* 1999; 56 (3):225–232.
Siniarska A.: Rozwój biologiczny dzieci i młodzieży z kilku wybranych regionów Polski na tle warunków życia rodziny i pewnych cech biologicznych rodziców. *Studies Hum Ecol* 1994: supl.1:89–194.
Snethen G., Puymbroeck M.V.: Girls and physical aggression: Causes, trends and intervention guided by Social Learning Theory. *Aggress Violent Beh* 2008; 13:346–354.
Spear L.P.: The adolescent brain and age-related behavioral manifestations, *Neurosci and Biobehav R* 2000; 24:417–463.
Torsheim T., Currie C., Boyce W., Kalnins I., Overpeck M., Haugland S.: Material deprivation and self-rated health: a multilevel study of adolescence from 22 European and North American countries. *Soc Sci Med* 2004; 59:1–12.
Wang L.Y., Chyen D., Lee S., Lowry R.: The association between body mass index in adolescence and obesity in adulthood. *J Adol Health* 2008; 42(5):512–518.
West P.: Health inequalities in early years: is there equalization in youth? *Soc Sci Med* 1997; 44(6):833–58.
Zarzycki J.: Dojrzewanie płciowe i jego zaburzenia. In: M. Pawlikowski (Ed.) *Zarys endokrynologii klinicznej*. 1992. Warszawa: PZWL.
www.gios.gov.pl

Wiesław Osiński, Janusz Maciaszek, Robert Szeklicki

# Physical fitness of adolescents in the Wielkopolska province versus Poland's population

**Abstract:** The study objective was to determine and assess the physical fitness level of children and adolescents aged 10 to 18 years from the Wielkopolska versus the total Poland's population. The physical fitness tests were carried out on a randomly chosen population of 2,269 boys and 2,288 girls aged 10 to 18 years. In the physical fitness assessment six tests from the Eurofit test battery were used, estimating flexibility, explosive power, static strength, trunk strength, functional strength, running speed/agility. The Cooper Test estimating cardio-respiratory endurance was also used.
Centile charts were constructed and the physical fitness level was assessed in comparison to Poland's population with respect to the subjects' gender and calendar age categories.
In comparison with the all-Poland population, older boys from the Wielkopolska tend to have a dramatically lower physical fitness level within the majority of other physical fitness elements and studied age groups. The physical fitness level of girls, especially in higher age categories (17–18 years of age), can be described as satisfactory. The causal explanation of the observed tendencies in the development of physical fitness of children and adolescents requires additional research of the gathered data. A general research analysis shows that a special focus should be placed on support the physical fitness developing of boys. The physical fitness assessment of a population may only serve as an introduction to the evaluation of individual needs of boys and girls that shall be analysed in relation to somatic development level and real health criteria.

**Key words:** physical fitness, boys and girls, centile charts, norms

## Introduction

Physical fitness is a commonly used but variously defined term. Irrespective of the detailed understanding of the term, physical fitness is nowadays regarded as a component of a positive health assessment and its right level is considered as a desirable condition for every human being. Whether we speak about the physical fit-

ness of a child or of an adult, of an office worker or of a sportsman, it always improves the wellbeing and the quality of life.

Physical fitness is often associated with mastering a significant number of physical skills and with an efficient cardiovascular, respiratory, secretory and thermoregulatory system. Physical fitness is furthermore identified with the biological value and physical condition of a human being, and the connection between physical activity and good health is commonly accepted [Franks 1989; Bouchard and Shephard 1994].

In relation to children and adolescents, physical fitness is considered as one of the assessment criteria of a harmonious development. In this assessment the motor performance perspective on physical fitness is gradually abandoned and replaced by more health-related components. That was the approach of the authors of this study while selecting physical fitness tests. The tasks that have been chosen describe the components mentioned in typical classifications of health-related fitness (H-RF) [Bouchard and Shephard 1994; Skinner and Oja 1994; Wuest and Bucher 1996].

An important criterion for the choice of fitness tests was the possibility of comparing the obtained results, especially with the all-Poland population. The same applies to the quality assessment of the physical fitness. It shall be stressed that the assessment of a population comprising various calendar age categories serves exclusively as a basis for a general summary of test results or for social and educational policy guidelines. Physical fitness testing is an important pedagogical tool in the fitness education of children and adolescents. Keeping the ADOPOLNOR research project in view, the authors have been encouraging physical education teachers during direct trainings to adopt this approach to physical fitness tests. The physical fitness diagnosis shall be an occasion to inform about the essence and importance of particular components of physical fitness, about the ways to support the development and to sustain the achieved level over the entire course of life. The overall goal should be health promotion and concern about the functional wellbeing, and the applied physical fitness tests should primarily assess the elements closely related to health [Oja, Vand, Tuxworth 1995]. It has been assumed that promoting life-long physical activity and lifestyle change is a much more crucial task than merely indicating where an individual stands against a background of the entire population.

The objective of this study was to determine and assess the physical fitness level of children and adolescents from the Wielkopolska region aged 10 to 18 years in reference to Poland's population as a whole.

## Material and methods

The physical fitness tests have been performed on a representative, randomly selected population of 2,269 boys and 2,288 girls, aged 10 to 18 years and living in villages, towns and cities of the Wielkopolska region of Poland. The middle value of

calendar age ranges were integral values which meant that for example a group of 10-year-olds comprised children aged 9.50 to 10.49 years on the test day. The detailed number of studied boys and girls in particular calendar age ranges has been presented in Table 1. The table presents a maximum number of subjects taking part in the physical fitness tests. A lower number of subjects in certain fitness tests was caused by the lack of possibility of all subjects taking part in a particular test (medical obstacle to do a physical exercise, injury or another indisposition during the test period).

Table 1. Number of examined boys and girls in particular age categories

| Age | Boys | Girls |
|---|---|---|
| 10 | 127 | 117 |
| 11 | 276 | 274 |
| 12 | 264 | 302 |
| 13 | 255 | 276 |
| 14 | 252 | 270 |
| 15 | 291 | 275 |
| 16 | 254 | 310 |
| 17 | 257 | 189 |
| 18 | 293 | 275 |
| Total | 2269 | 2288 |

The physical fitness assessment was always performed by a physical education teacher using seven separate tests reflecting the level of basic factors of physical fitness (Table 2). Six tests have been taken from the Eurofit test battery [Eurofit 1993]. For the cardio-respiratory endurance assessment the well known and frequently used Cooper Test was applied [Cooper 1978]. In test selection, the focus has been primarily placed on health-related fitness elements.

Table 2. Investigated physical fitness factors and names of tests applied

| Item | Physical fitness factor | Name of test |
|---|---|---|
| 1 | Flexibility | Sit and reach |
| 2 | Explosive power | Standing broad jump |
| 3 | Static strength | Hand grip |
| 4 | Trunk strength | Sit-ups |
| 5 | Functional strength | Bent arm hang |
| 6 | Running speed/agility | Shuttle run: 10×5 m |
| 7 | Cardio-respiratory endurance | Cooper Test: 12 minutes running |

The teachers carrying out the physical fitness tests had taken part in a special training organized in the project's seat and conducted by the employees of the University School of Physical Education in Poznań, the authors of this work. The training included a multimedia presentation explaining how to perform the measurements and record results.

Below there is a detailed instruction given to the teachers on what equipment was required, how to carry out the tests and how to record the results. It also provides a description of the applied research methods. The results were entered in special test forms which apart from fitness tests results also included the following information: subject's name, surname, date of birth, gender, school, class; test start and end date, and tester's name, surname and signature. It was recommended that the measurements should be carried out very carefully, with a total commitment of the physical education teacher and all participating pupils, and fully in true with the instruction. In case of any doubts, teachers conducting the tests could contact the research supervising team at the University School of Physical Education in Poznań.

**Instruction on how to carry out the measurements of children's and adolescents' physical fitness as a part of a research project (ADOPOLNOR). "At the doorstep to adulthood: adolescent health and quality of life in a variety of socio-economic backgrounds".**

## General guidelines

1. It requires on average approximately four to five physical education classes to carry out all physical fitness tests in a given class by one physical education teacher. In practice, due to absences, sick leaves or indisposition of particular pupils, the physical fitness tests may require up to seven physical education classes.
2. It is recommended that according to Table 2 – if possible – on the first day of the research tests 1, 2 and 3 are performed and on the second day tests 4, 5 and 6, on the third day the pupils are being prepared for the Cooper Test and on the fourth day test 7, the Cooper Test, should be conducted.
3. Irrespective of how many days it will take to conduct all tests, it is recommended that the given order is kept (Table 2).
4. All Eurofit tests listed below should be carried out at a indoor gym. Exception is the Cooper Test which should be conducted in a sports field or in another open area which fulfills relevant requirements.
5. The subjects participate in the tests wearing sports clothes (loose, short or long trousers that do not restrict movement, a t-shirt) and barefoot (apart from the Cooper Test).
6. In order to minimize the risk of injuries, it is recommended to conduct an approximately 10-minutes long warm-up of a medium intensity on each day, prior to the measurements. The warm-up should comprise basic exercises for the main joints and muscle groups (apart from two exercises which are compulsory before the trunk flexibility test and which are described below).
7. Test results cannot be used as a basis for any school marks. It would be, however, recommended to – if possible – individually discuss test results with pupils (as well as with their parents), providing them with advice on a suggested physical activity programme, a sensible diet or a healthy lifestyle!

### 1. Flexibilty (sit and reach)

*Equipment and facilities*. Sit and reach box borrowed from organizers with a centimetre scale (in the longitudal axis) set in such a way that the 15 cm value on the scale is at the height of the sitting subject's foot support (at the level of the outer edge of the side wall of the bench). A ruler (approximately 20 cm long) is placed loosely on the surface of the box, at right angles to its longitudal axis and it should be moved very calmly with fingers during the forward bend.

*Procedure*. Before the actual test, each subject should perform two static stretching exercises shown in Figure 1. Each of these exercises should be done twice per each lower limb during approximately 20 to 30 seconds.

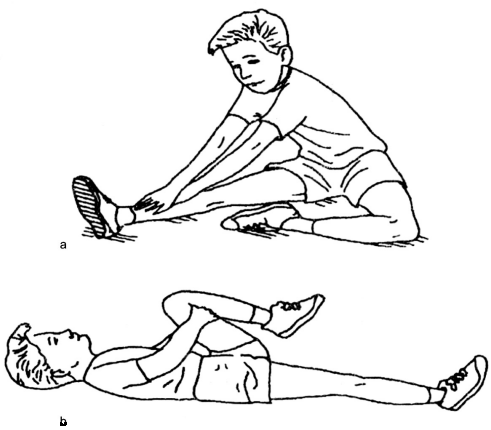


Fig. 1. Warm-up stretching exercises before sit and reach test

*Start position*: the subject sits on the floor facing the box with legs stretched out straight ahead and joined, knees fully stretched out, feet resting on the side wall of the box, arms raised upwards.

*Task*: the subject bends slowly forward and reaches with their hands forward as far as possible moving the ruler calmly over the surface of the box. When the maximum distance is reached, the subject should stop the movement for 2–3 seconds. During the bend the teacher can gently (not allowing even the slightest bend of knees but not pushing the pupil's knees to the floor either) hold the pupil's knees (Fig. 2).

If the subject's lower limbs are even minimally bent at knee joints, the result should not be recorded and the test should be repeated.

Result. The edge of the shifted ruler which is closer to the subject determines the reach of the forward bend. If the ruler is placed diagonally on the scale of the box, an average value should be determined, i.e. two extreme values should be added and divided by two. The subject performs the test twice and the better score

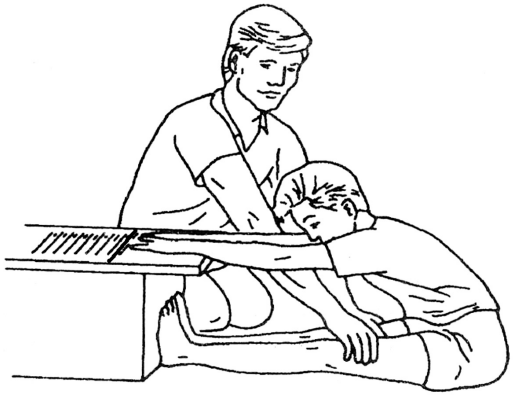


Fig. 2. Flexibility testing method (sit and reach)

is collected. When the subject does not reach the 0 (zero) point on the centimetre scale (not to be mistaken with the foot support point, where the value of 15 should be entered), the score of 0 (zero) should be entered in the test form.

### 2. Explosive power (standing broad jump)

*Equipment and facilities*. Test should be carried out on a non-slip surface and two or three landing mats, connected as firmly as possible. A measuring tape should be placed along the mats so that 0 (zero) is placed exactly at the take-off line.

*Procedure*. Start position: the subject stands barefoot with feet slightly apart (not exceeding hip-width) behind the take-off line.

*Task* (Fig. 3): the subject bends the knees simultaneously swinging the arms down and backwards, then jumps forward as far as possible by pushing themselves with their legs against the ground and swinging the arms forward. A two-foot landing is used, keeping the feet (the vertical position) at the landing point. The teacher should ensure (especially in the lower classes) that pupils have the ability to perform a standing broad jump. If this is not the case, this ability should be practiced prior to the test. A frequent mistake is a take-off at a wrong angle (upward jump or too flat jump).

*Result*. Using the measuring tape along the landing mats, the teacher determines the jump length (within an accuracy of one centimetre). The reading point is the subject's heel which is closer to the take-off line. The test is done twice and only the higher score is entered in the test form. If the subject falls or stumbles after the jump, or moves their foot (feet) after landing, the jump has to be repeated.

### 3. Static strength (hand grip)

*Equipment and facilities*. Proven handgrip dynamometer. It is recommended to use solely a dynamometer with a handle adjustable to the subject's hand width, borrowed from the organizers.

*Procedure*. Start position: the subject stands with feet slightly apart, holding the dynamometer in the fitter hand (chosen by the subject). Arm rests along the side

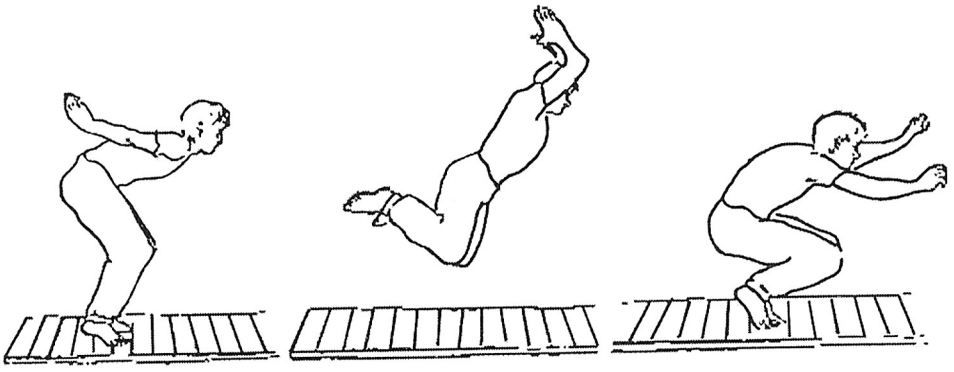


Fig. 3. Explosive power testing method (standing broad jump)

Fig. 4. Static strength testing method (hand grip)

of the torso so that neither the hand or the dynamometer touches the body or any other object. The second arm rests comfortably along the other side of the torso.

*Task* (Fig. 4): squeezing dynamometer during a short period (2–3 seconds) with a maximal strength.

*Result*. The test is performed twice, using the same hand chosen by the pupil. The result is read from the dynamometer scale and the better one is entered in the test form within an accuracy of 0.5 kg.

### 4. Trunk strength (sit-ups)

Equipment and facilities. Hard floor mat, stopwatch, partner to hold feet.

Procedure. Start position: the subject sits on the floor mat, legs bent at the knee joints at right angles, feet hip-width apart, fingers interlocked behind the head and elbows touching the knees. The partner sits in front of the subject (Fig. 5) and holds their feet down so that the whole sole of the feet touches the ground during the entire test.

*Task*: on the command of "go", the subject moves from sitting to lying position as fast as possible during 30 seconds (touching the mat with their shoulders) and returns to the sitting position holding the elbows forward in order to touch the knees with them. The extreme positions should be monitored closely: while lying, it is enough to touch the mat with shoulders and while sitting it is the elbows (and not the head!) that touch the knees (Fig. 5).

*Result*. In the test form the number of correct, full-cycle sit-ups performed during 30 seconds should be entered. For example, 20 correct sit-ups give the score of 20.

### 5. Functional strength (bent arm hang)

*Equipment and facilities*. Horizontal bar fixed at a level that allows the tallest subject to perform a full hang; floor mat; stopwatch; partner.

*Procedure*. Start position: the subject stands on the floor mat beneath the bar. The partner (or the teacher) supports the subject by holding their hips so that the bar can be grasped using an overhand grip. Arms should be shoulder-width apart and bent at the joints. The chin should be above the bar but not resting on it (Fig. 6).

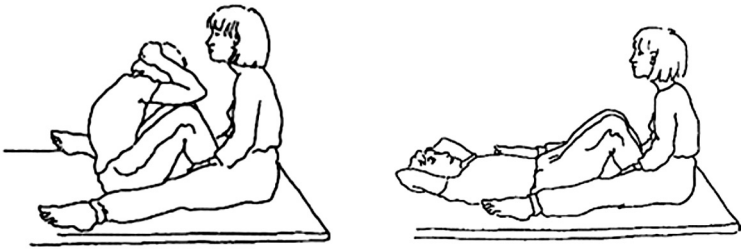


Fig. 5. Trunk strength testing method (sit-ups)

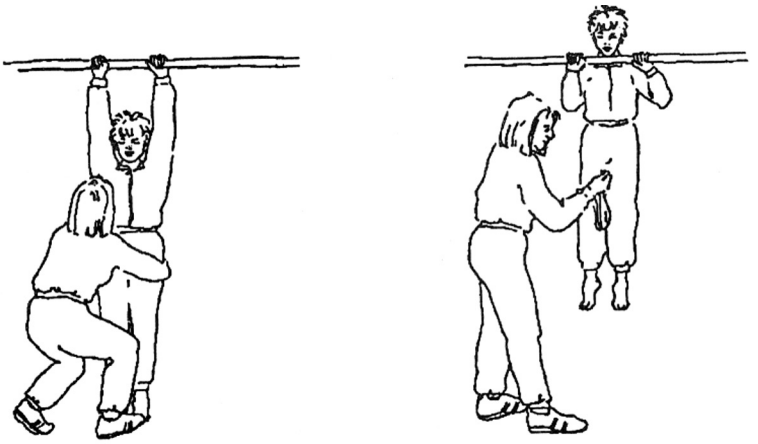


Fig. 6. Functional strength testing method (bent arm hang)

*Task*: the subject should keep the position as long as possible (Fig. 6). On the timekeeper's command of "go" (it is not advisable to delay the "go" command), the partner fully releases the subject who maintains the hanging position with chin above the bar as long as possible. It is not allowed to support oneself against the bar!

*Result*. The test is done once. The time of the correct hanging position is entered in the test form (within an accuracy of 0.1 second) which is measured from the moment of release and the "go" command until the moment when the subject's eyes are below the bar.

### 6. Running speed/agility (shuttle run: 10×5 m)

*Equipment and facilities*. Flat and non-slip surface, stopwatch, measuring tape, chalk, 4 gum marker cones. Two parallel lines are drawn on the floor, each 120 cm long, 5 m apart. Marker cones are placed at the ends of the lines.

*Procedure*. Start position: standing position behind the start line (Fig. 7).

Task: on the command of "go" the subject runs as fast as possible to the second line 5 metres away and returns, crossing the line every time with both feet. The five-metre distance has to be run 10 times. It is important to make sure that the subjects do not brace their hands against the ground for support during the turn.

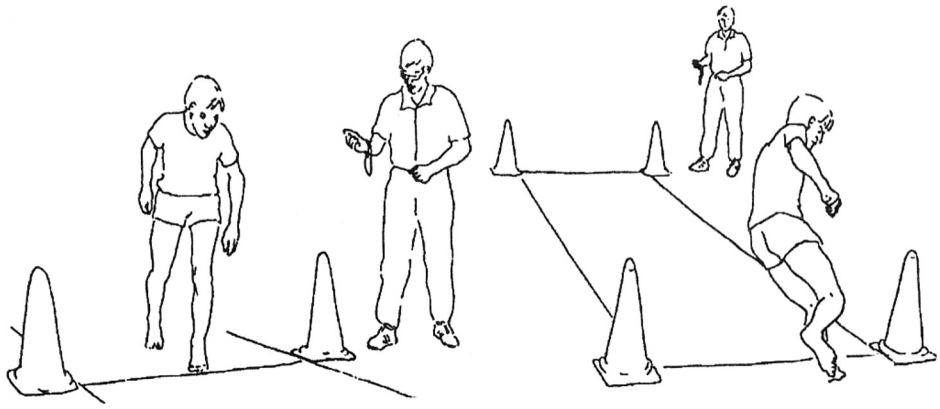


Fig. 7. Running speed/agility testing method (shuttle run: 10 × 5 m)

*Result*. The test is done once. The time necessary to cover 10 full stretches is entered in the test form (within an accuracy of 0.1 second).

### 7. Cardio-respiratory endurance (Cooper Test)

*Equipment and facilities*. It is recommended that the test is conducted on a 400-metre oval running track after checking the lap length. If no such track is available, a closed oval- or ellipse-shaped route should be marked out where one lap should not be shorter than 250 meters and with 50-metre stretches marked to make it easier to estimate the covered distance. Stopwatch, whistle.

*Procedure*. Start position: standing position behind the start line.

*Task*: the subject should run as far as possible during 12 minutes. The studied pupils should enter the test in sportswear, including sports shoes.

The test can be carried out in groups of a maximum of around fifteen participants. It is advisable that the subjects are divided into two equal groups and pairs are set in which one pupil performs the exercise and the second one counts the laps. During the run the subjects are informed about the time left until the end of the exercise. Once 12 minutes have passed, the whistle blow signals the end of the run. The subject should stop and stay on the spot until the tester determines the runned distance. Depending on the advancement level, the subject can do the test by choosing to run, run/walk or walk.

An objective measurement can only take place if the subject enters the test well motivated, covers the distance at a relatively even pace and finishes the attempt significantly tired (without sparing themselves).

The test can be carried out only in such places where there is a possibility to measure the length of the runned distance. The profile of the route and its precise measurement have a considerable impact on the result (the more difficult route, the worse result). Therefore the most suitable place is a running track at an athletics stadium.

During the Cooper Test, it should be ensured that strength is distributed as evenly as possible throughout the whole distance. Fast speed at the beginning of

the run is particularly inadvisable. If the subject is exhausted, they can switch from run to walk. It is recommended that prior to the actual test, at least one day in advance, the subject should be informed about the goal of the test and perform a trial run so that the individual running speed can be determined.

*Result*. The Cooper Test is done once. In the test form, the covered distance during 12 minutes is entered within an accuracy of 10 metres.

Please note that carrying out the Cooper Test in an accurate manner requires an exceptionally careful planning of all organizational elements of the measurement and a full mobilization during the test. Special difficulties may arise when it is necessary to individually count the laps run by pupils who significantly left behind the rest of the subjects and who are therefore lapped on the route.

## Results

The presented results description constitutes a general summary of the research that has been pursued. The results have been analyzed only considering the subjects' categorization by gender and calendar age. It is planned to present the picture of the physical fitness of children and adolescents from the Wielkopolska region in separate works including the factors related to diverse social background, somatic characteristics or other functional features falling into the scope of the research project that has been carried out. The research results have been presented as centile charts (Fig. 8–14) which illustrate the results spread for particular physical fitness tests and calendar age categories, for boys and girls separately. The centile charts have been constructed using the LMS method. The calculations have been performed in the Biology of Human Development Department, the Institute of Anthropology, the Faculty of Biology at Adam Mickiewicz University in Poznań.

The obtained results of the physcial fitness of children and adolescents from the Wielkopolska region have been presented against the background of the total Poland's population by checking what perecentage of the subjects has achieved the results in the determined ranges of the results of children and adolescents in Poland (Tables 3–9). For the fitness tests from the Eurofit test battery the 100-point tables of physcial fitness of children and adolescents in Poland have been used [Dobosz 2010]. For each fitness test a five-point ordinal scale has been created – from low to high level of physical fitness. The range corresponding to a medium level of physical fitness comprises results of approximately 51% of all people from the all-Poland population and the range below the medium level and above the medium level in total includes results of 77% of people. For the Cooper Test assessment a five-grade scale has been used. It has been created by Pilicz et al. [2002] on the basis of research on the same all-Poland population.

## Discussion of results

The analysis of the research results (centile charts) of the physical fitness of children and adolescents from the Wielkopolska region in relation to calendar age of the subjects shows an expected, permanent tendency to increase and improve the scores with age. Only a slightly marked tendency towards stabilization, or even small decrease of the scores is characteristic for older girls. The best scores for static strength were achieved by 17-year-old girls (Fig. 10). In case of explosive power the best scores belonged to girls aged 16 to 17 years (Fig. 9) and in the level of cardio-respiratory endurance a decline in scores was observed already after 13–14 years of age (Fig. 14). Similar tendencies were not observed among boys.

The results of an all-Poland research from 1999 were used as a reference point in the physical fitness assessment. The results of this research, also based on the Eurofit test battery, have been published in the form of centile charts [Stupnicki, Przewęda and Milde 2003] and extensively discussed in the scientific monograph by Przewęda and Dobosz [2003]. The Cooper Test results were evaluated against standards determined by Pilicz et al. [2002]. At present, it is the only available and the most relevant comparative material. In 2010, however, another course of research conducted every decade on a randomly selected all-Poland population has been concluded, but the research results are not available yet. The conducted analysis focuses on the percentage spread of the subjects number in particular ranges of the five-grade norm scale. This assessment is supposed to show only population tendencies which occur in the structure and level of the physical fitness of children and adolescents. Therefore it cannot be applied in the context of individual assessments, for example from the health needs' perspective. In this regard, we should attempt to answer a difficult question: how much fitness does a good health demand? [Oja, Tuxworth 1995]. The focus should be placed on relevant criteria (practical goals) and not on static norms [Docherty 1996]. Due to the objective of this report and the limited length of the chapter, the advancement level of somatic development as well as the level of sexual maturity have not been taken into consideration. The comparison of the physical fitness level of children and adolescents in the Wielkopolska region and the all-Poland population indicates the occurrence of interesting phenomena. The picture of the physical fitness development of boys and that of girls are almost entirely disparate. In fact, the only similarity is the level of flexibility and functional strength (Table 3, 7), where both boys and girls who took part in the tests achieved lower scores than the all-Poland population in all age categories.

From 18.6% to 39.3% of boys represented a definitely low level of flexibility in individual age categories while only from 1.7% to 6.8% represented a high level (Table 3). Similarly, from 12.7% to 34.6% of girls demonstrated a low level of flexibility and from 4.1% to 10.4% of girls demonstrated a high level (Table 3). Particularly unfavourable results were recorded for 17- and 18-year-old boys and girls as in this age category the low flexibility level characterized as many as 38.4–39.3% of boys and 30.1–34.6% of girls. Flexibility and functional strength were only physi-

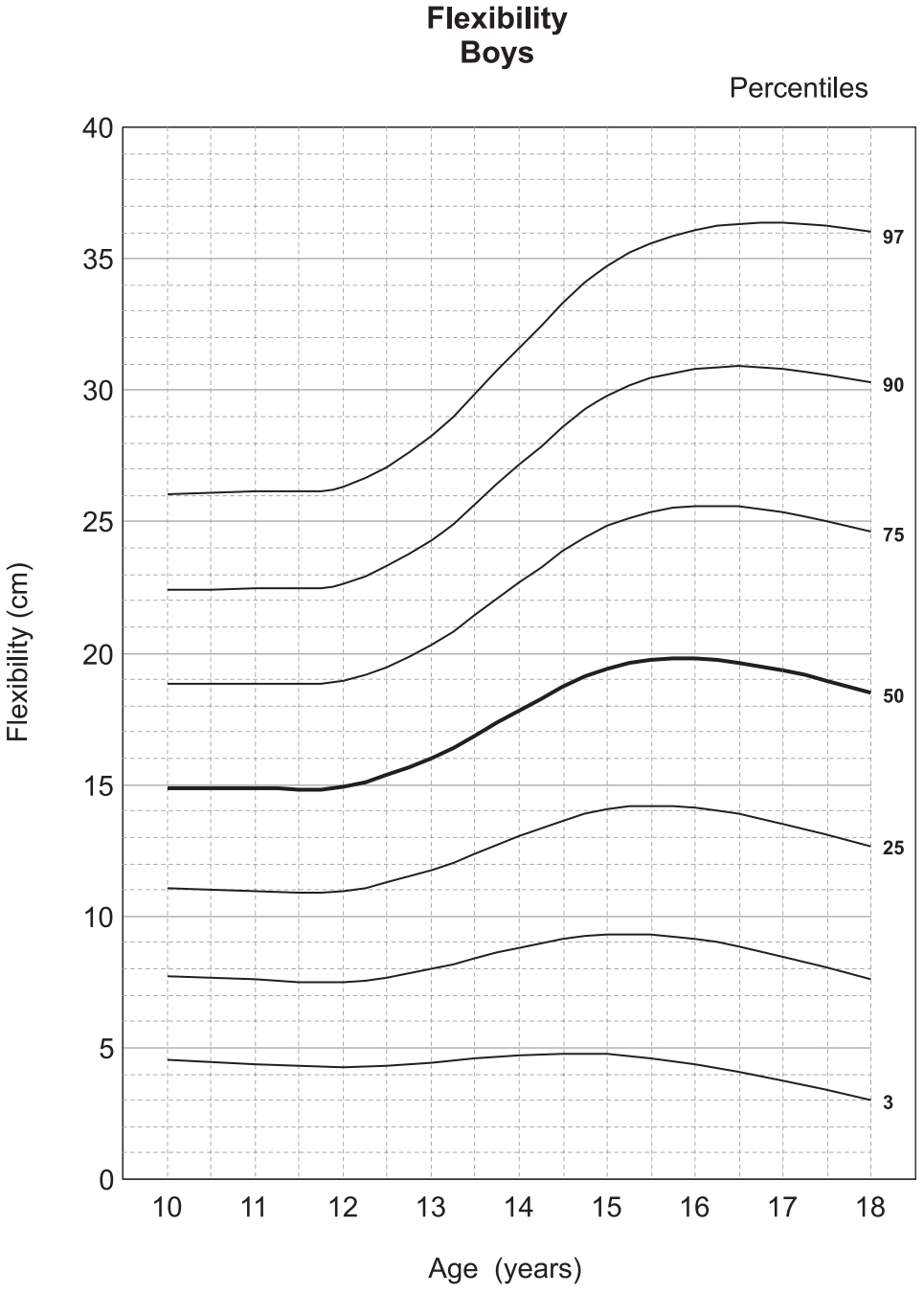



Fig. 8. Centile charts for evaluation of flexibility in boys by sit and reach test

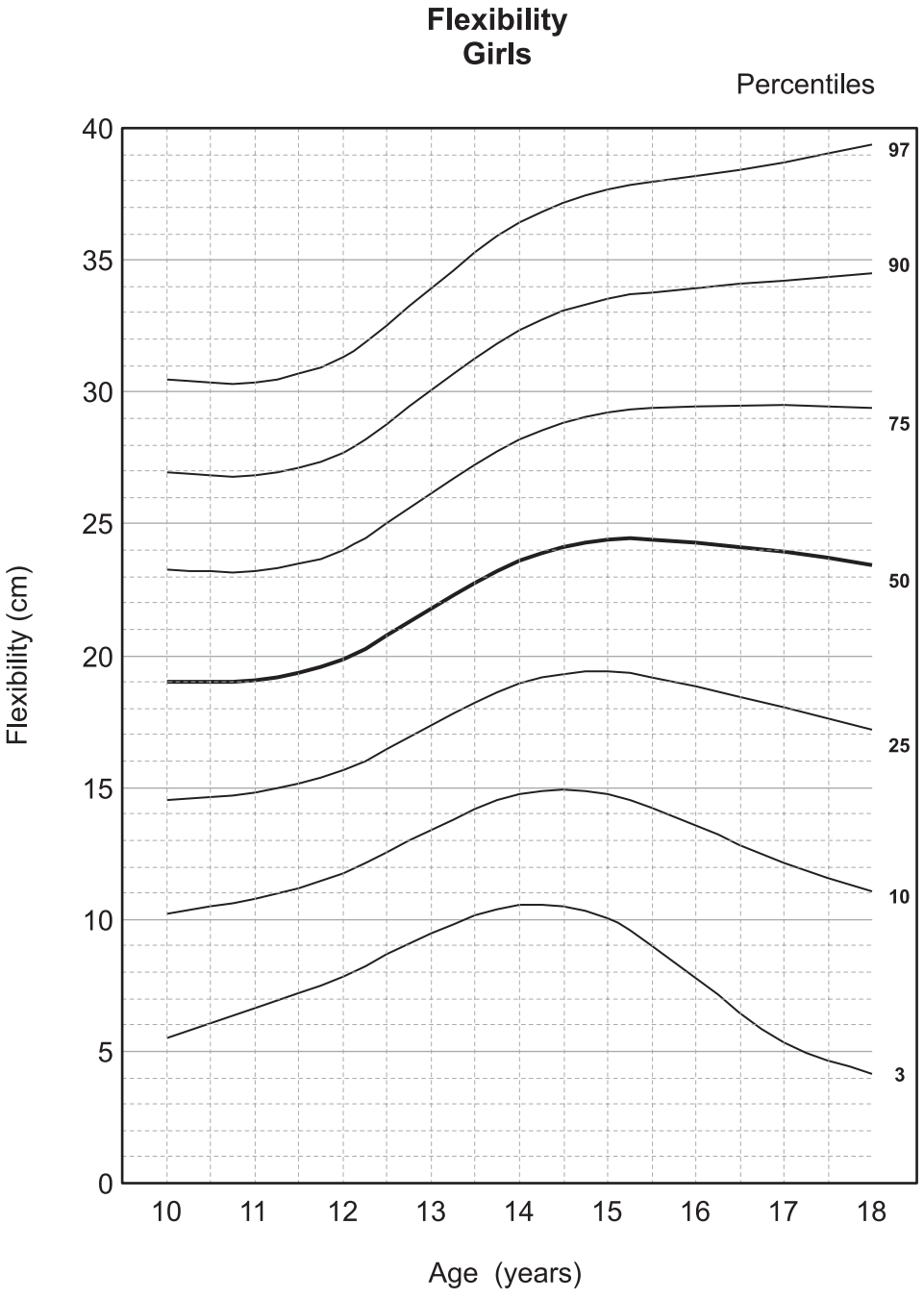



Fig. 8. Centile charts for evaluation of flexibility in girls by sit and reach test

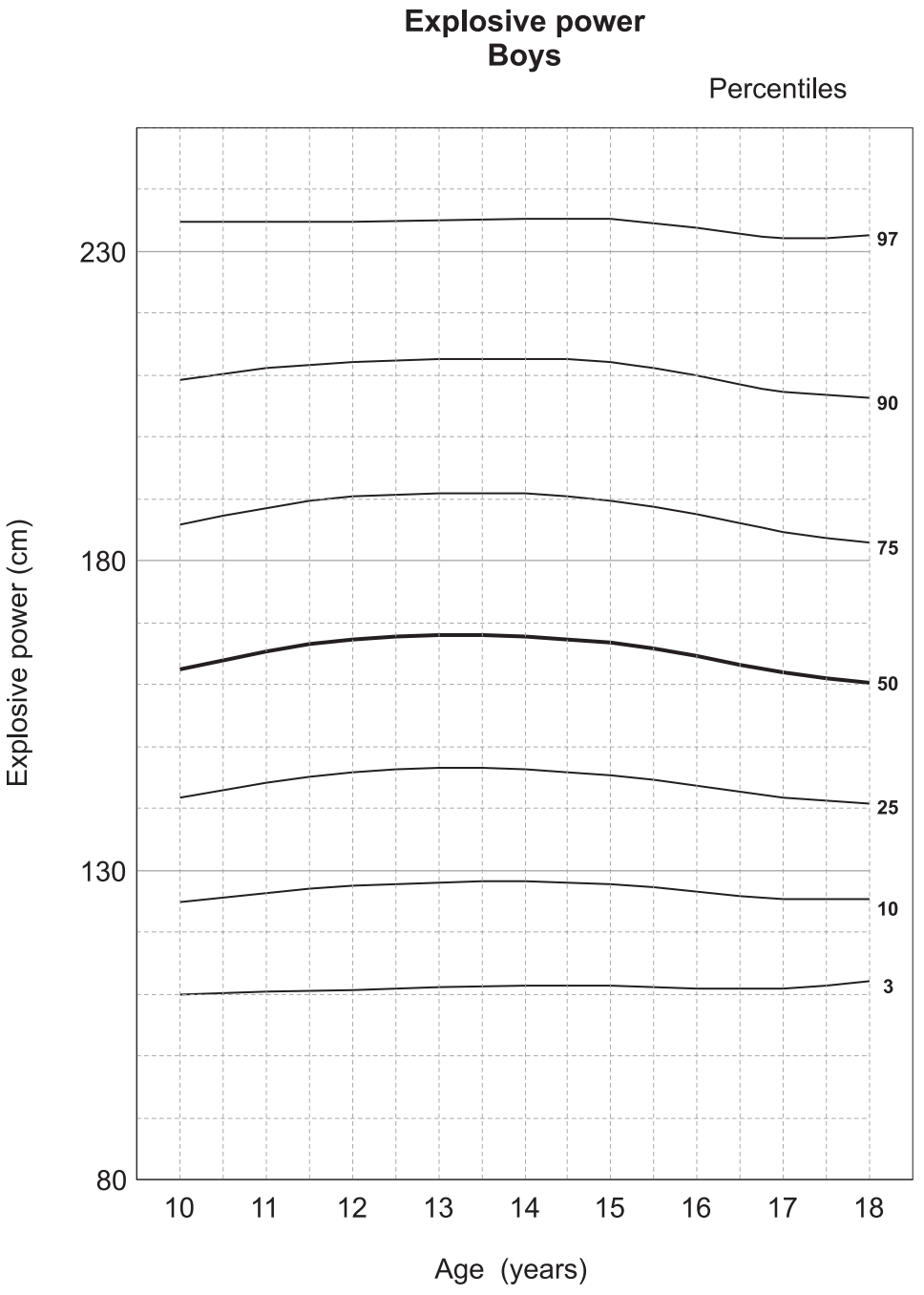



Fig. 9. Centile charts for evaluation of explosive power in boys by standing broad jump test

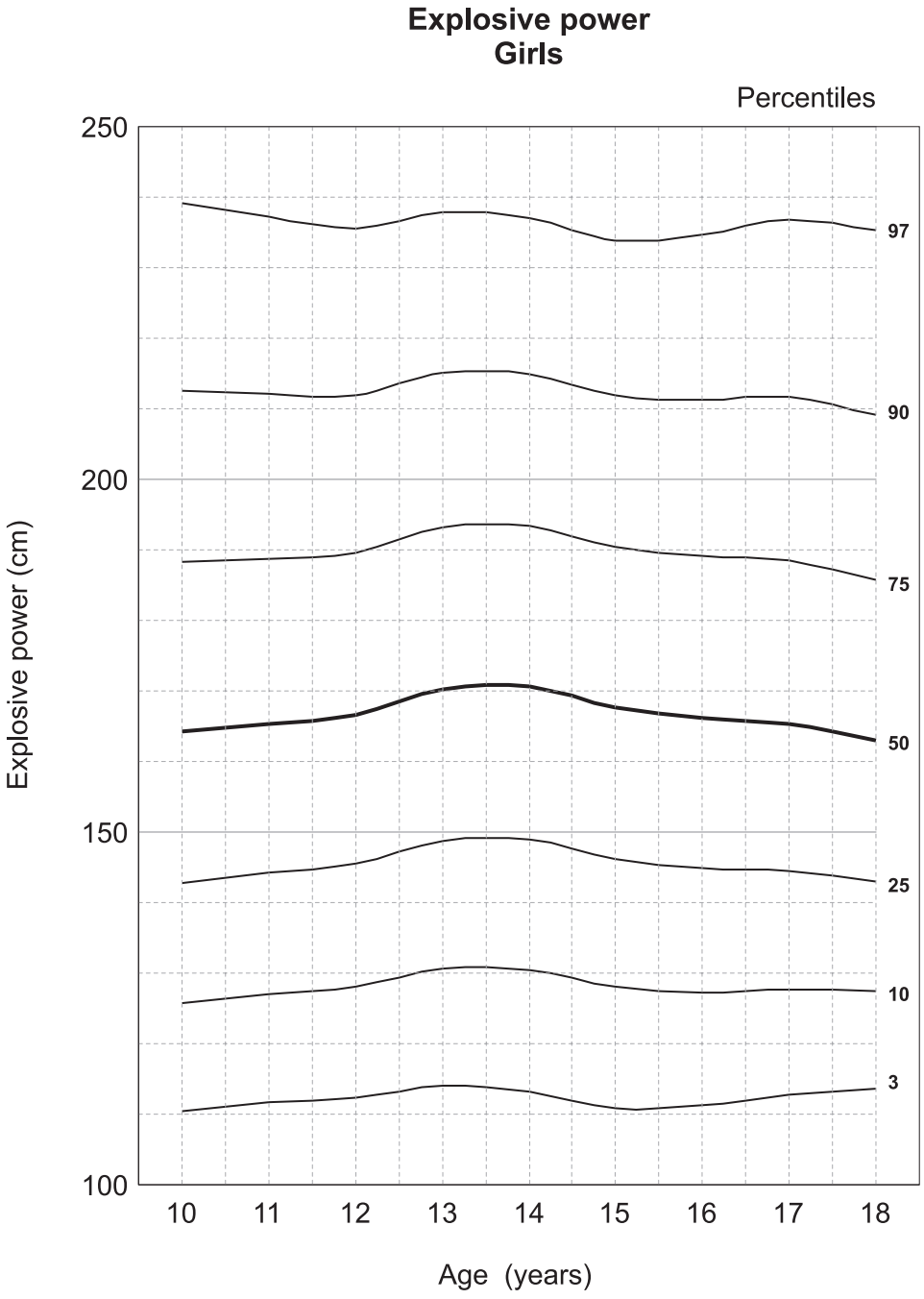



Fig. 9. Centile charts for evaluation of explosive power in girls by standing broad jump test

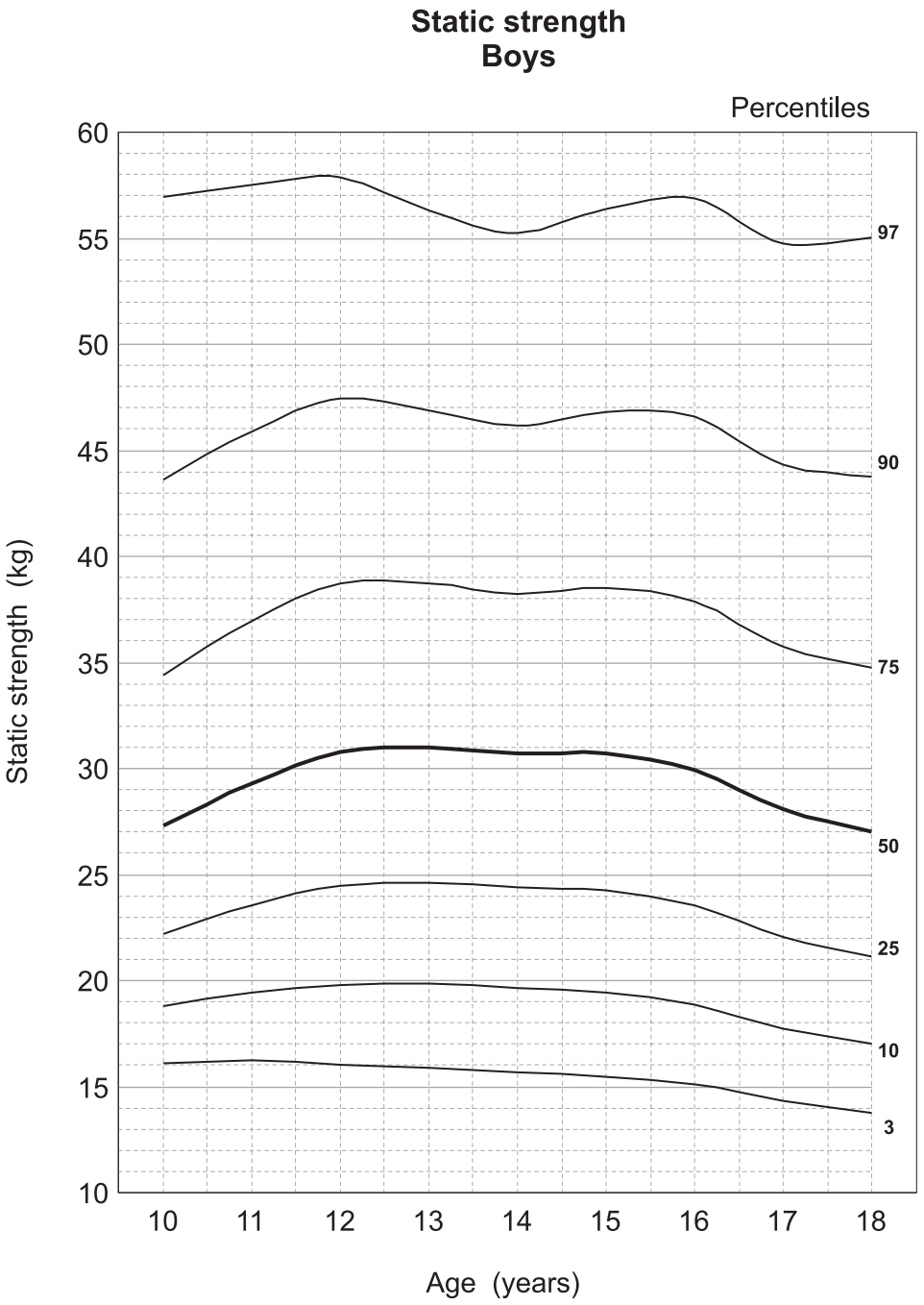



Fig. 10. Centile charts for evaluation of static strength in boys by hand grip test

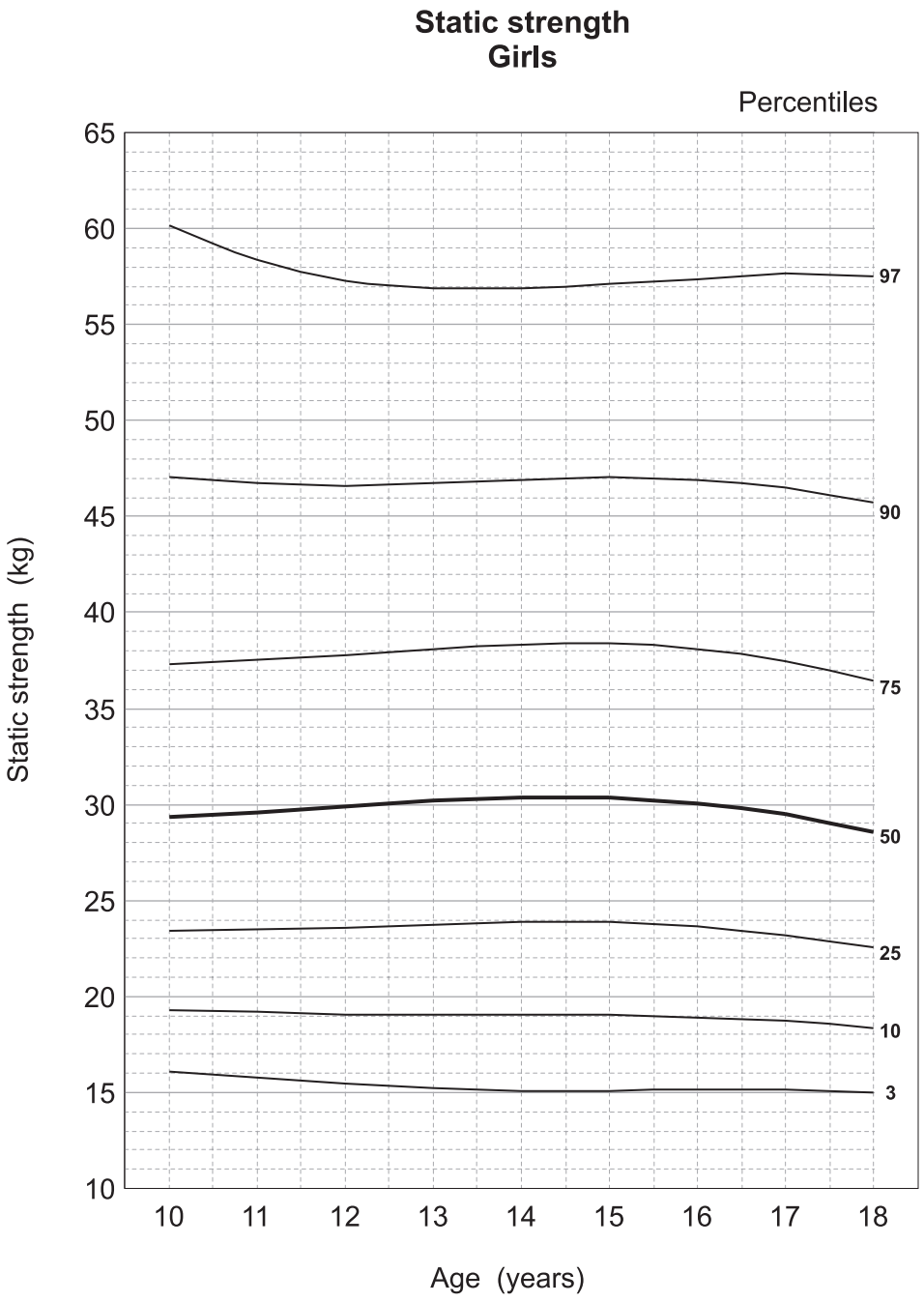



Fig. 10. Centile charts for evaluation of static strength in girls by hand grip test

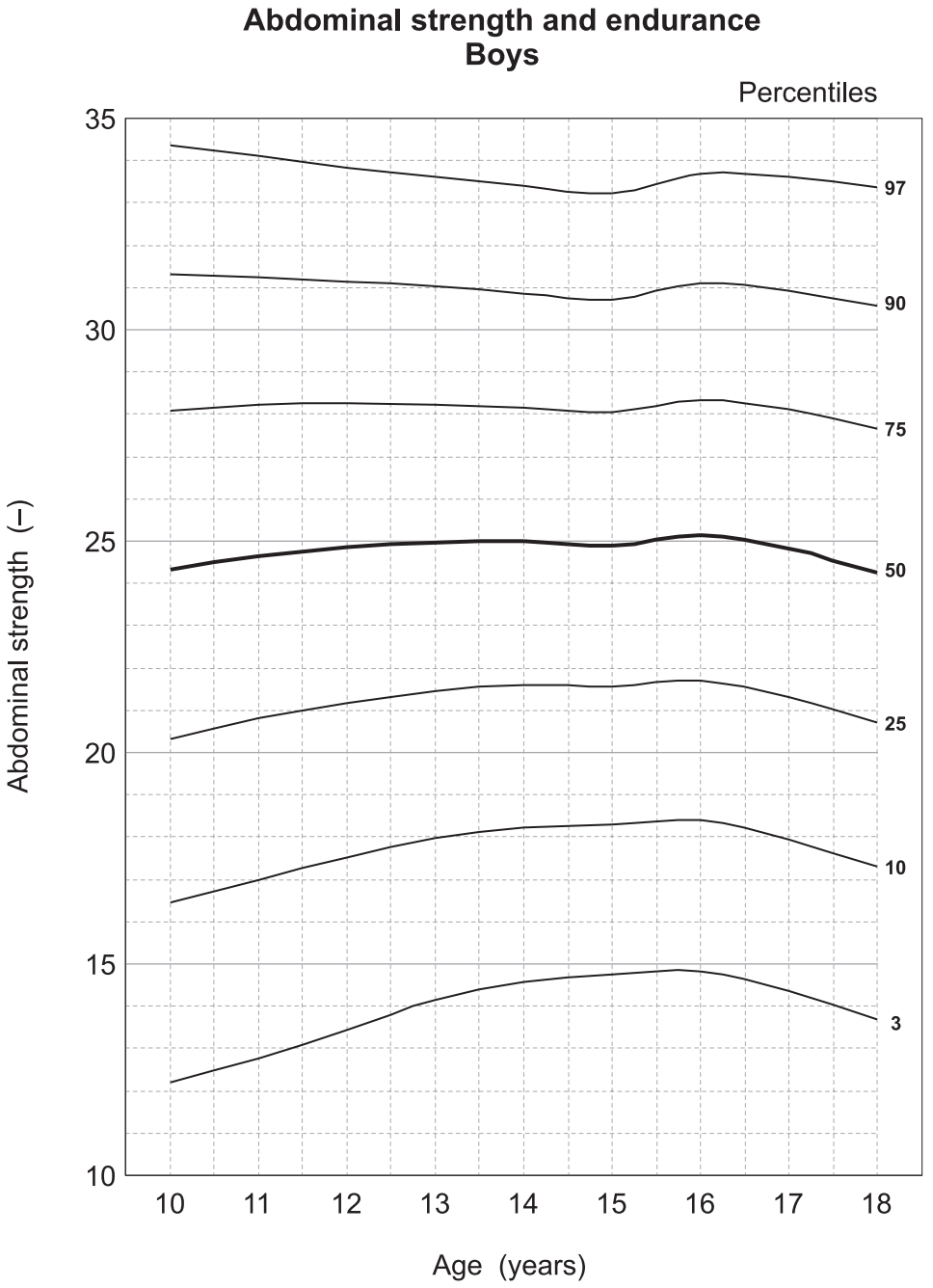



Fig. 11. Centile charts for evaluation of trunk strength in boys by sit-ups test

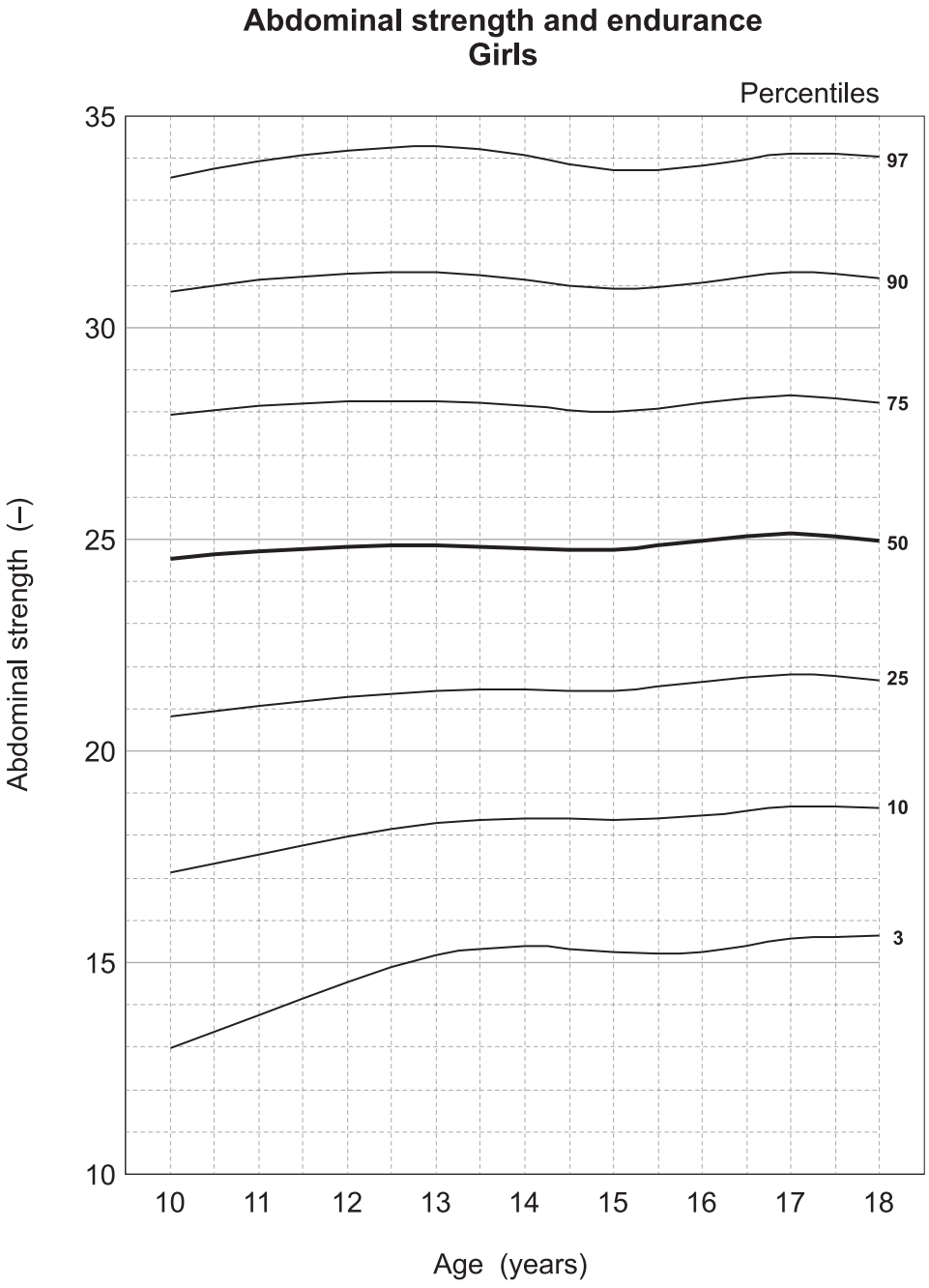



Fig. 11. Centile charts for evaluation of trunk strength in girls by sit-ups test

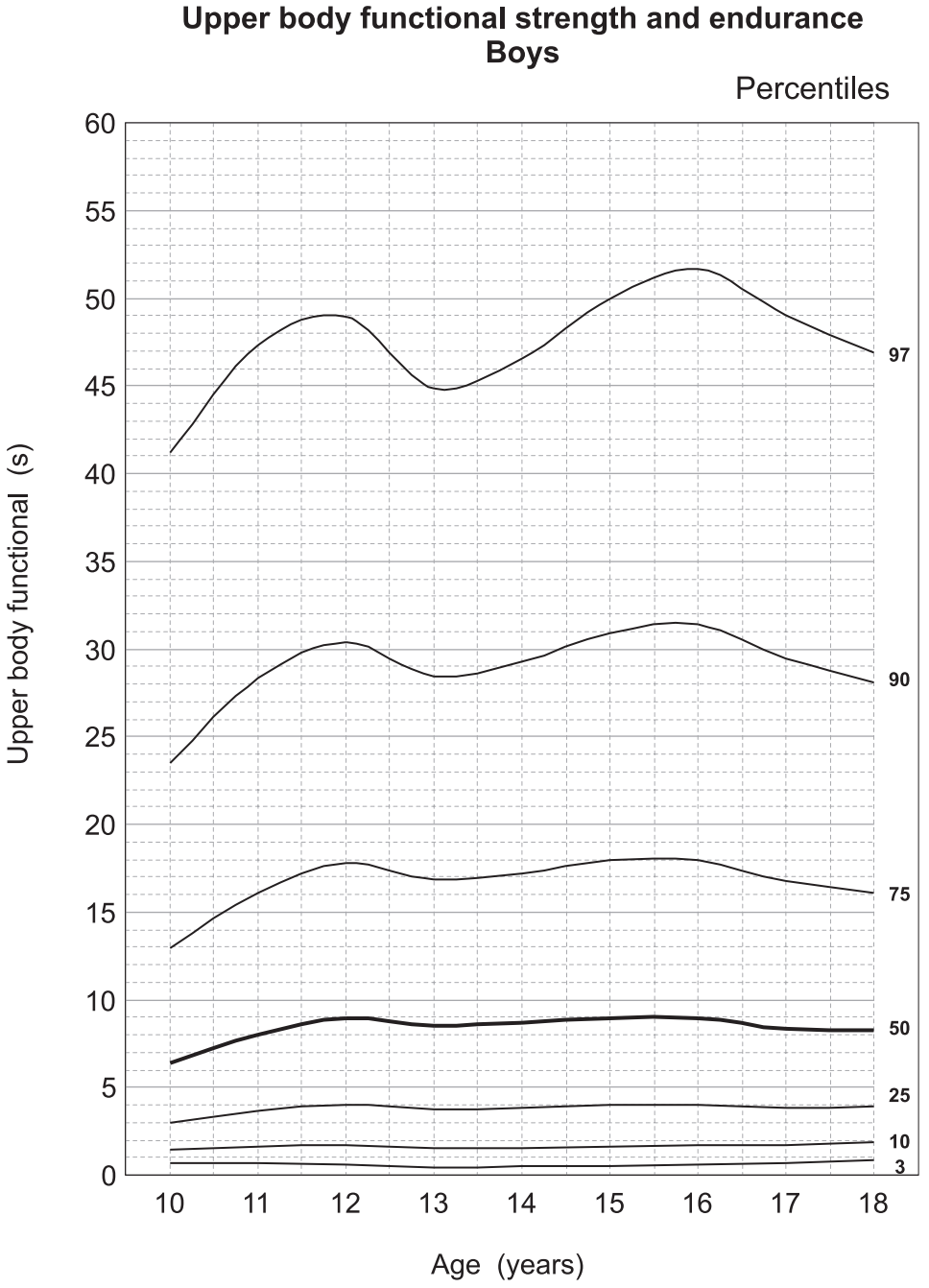



Fig. 12. Centile charts for evaluation of functional strength in boys by bent arm hang test

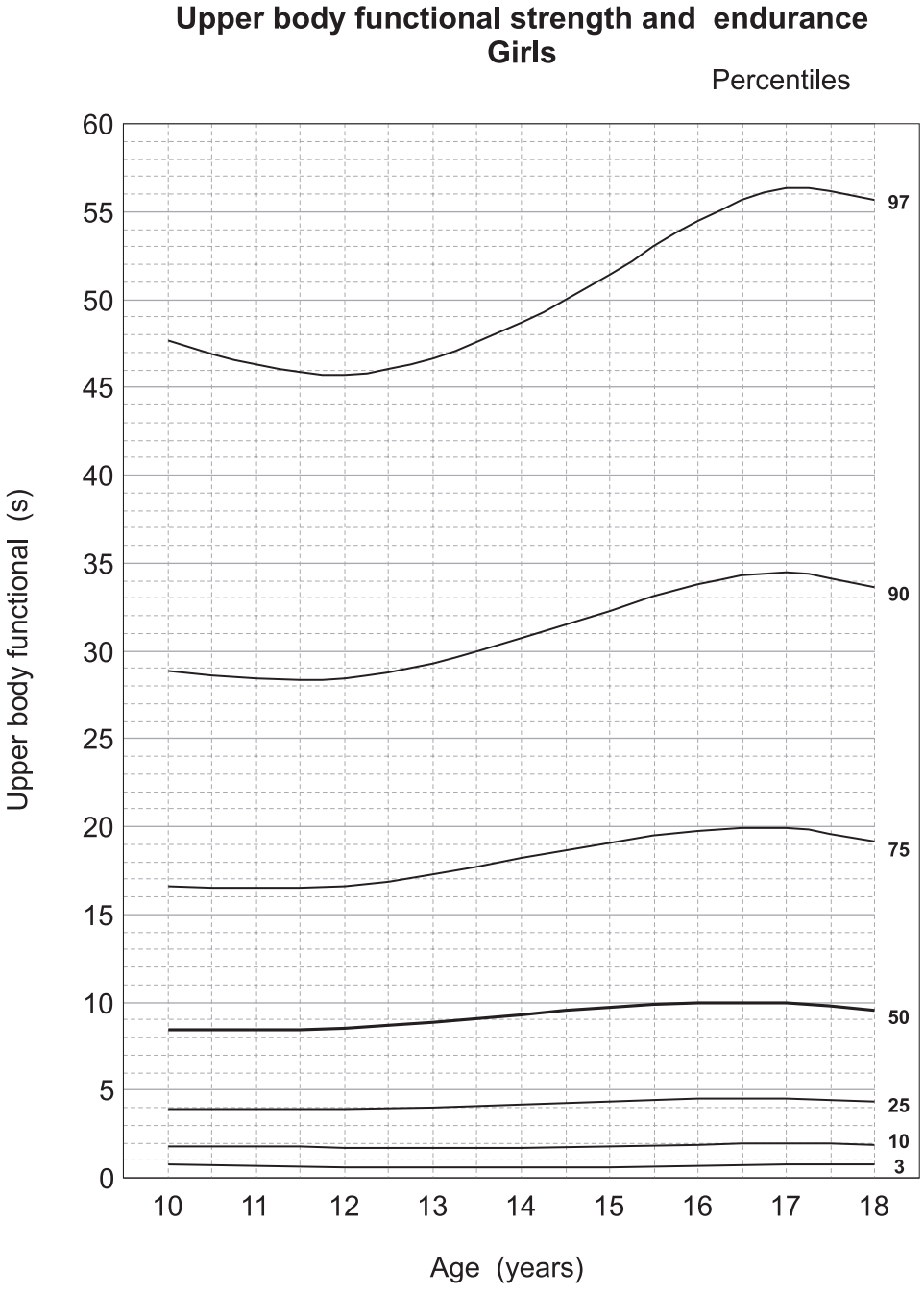



Fig. 12. Centile charts for evaluation of functional strength in girls by bent arm hang test

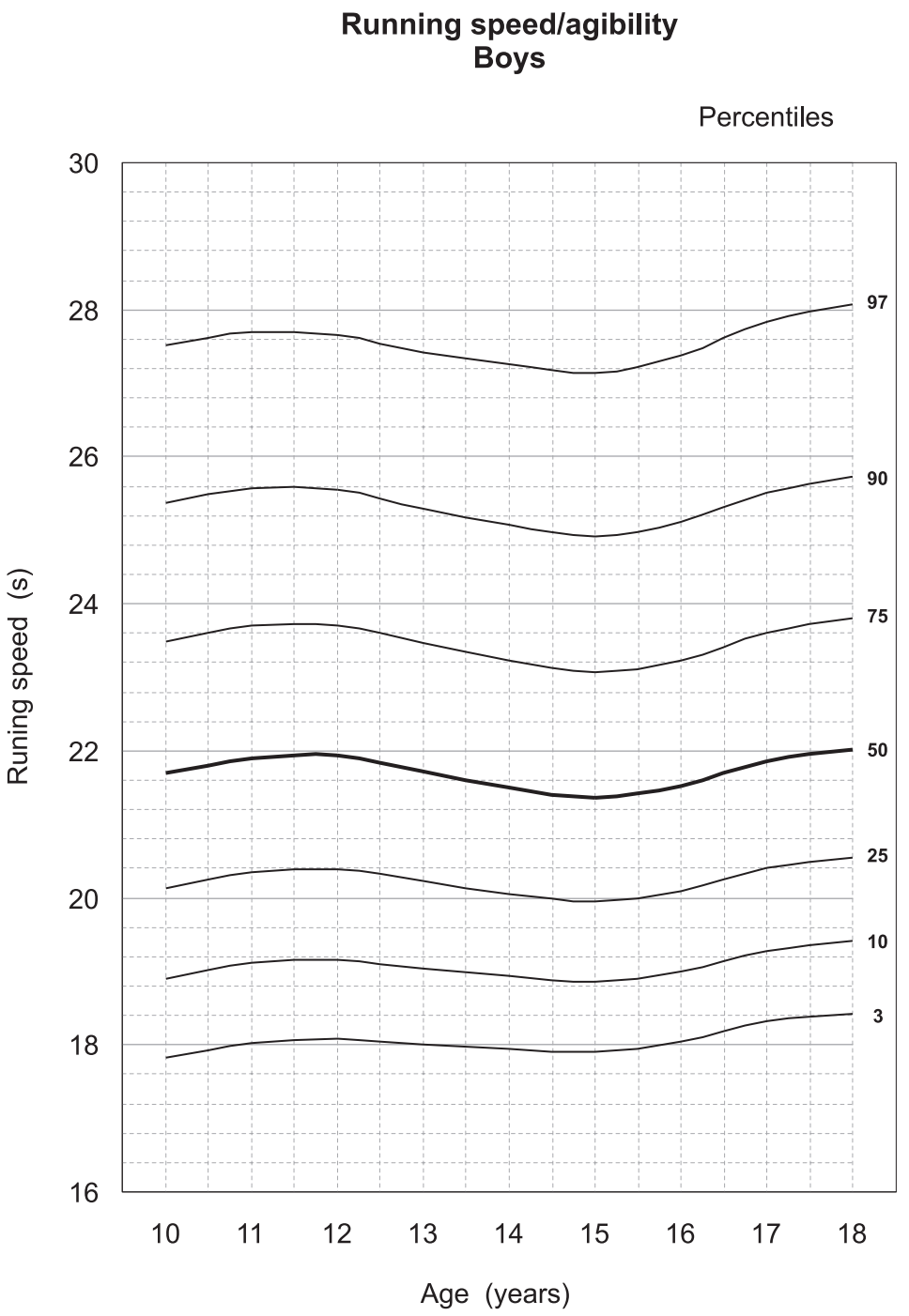



Fig. 13. Centile charts for evaluation of running speed/agility in boys by shuttle run: 10 × 5 m test

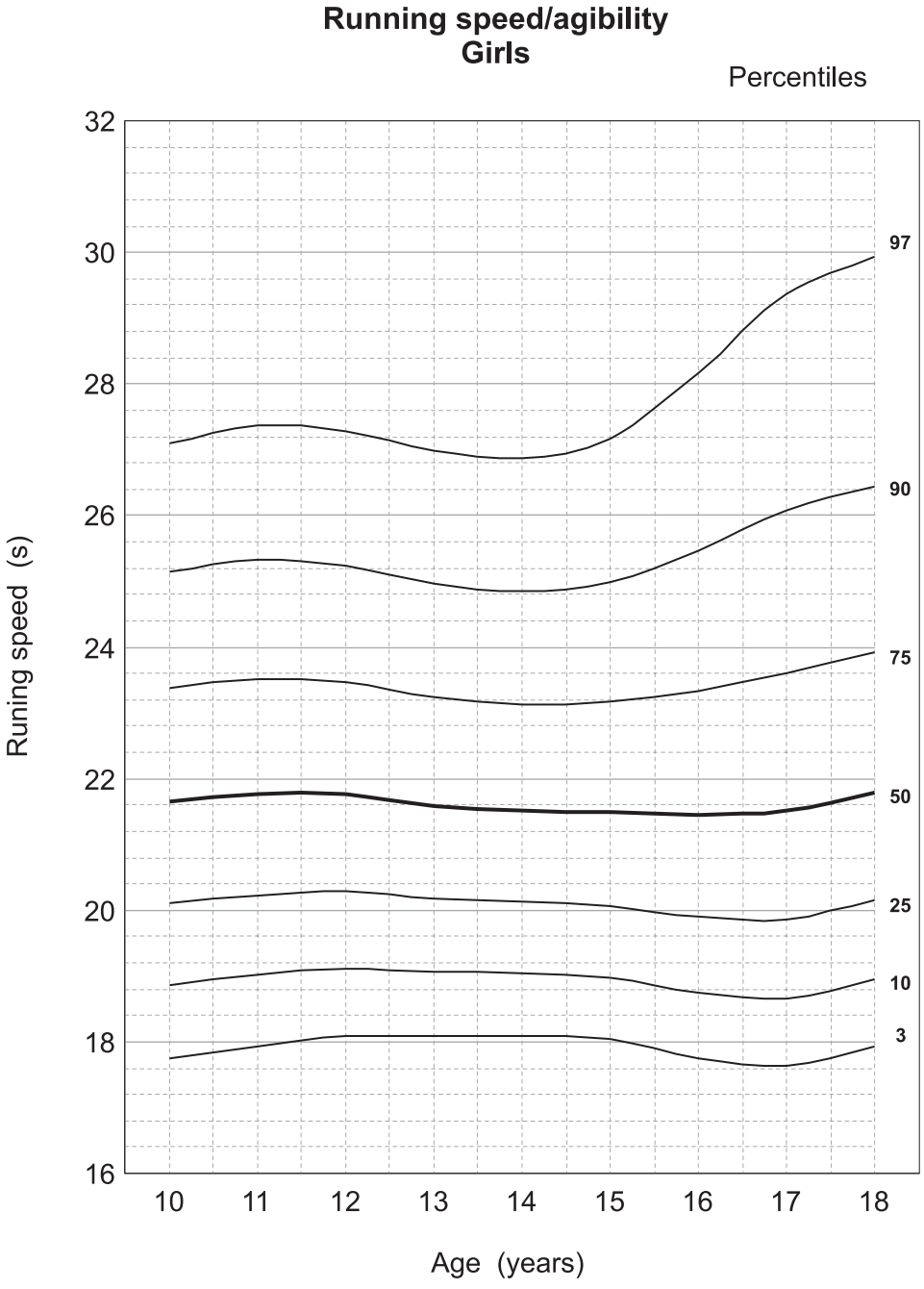



Fig. 13. Centile charts for evaluation of running speed/agility in girls by shuttle run: 10 × 5 m test

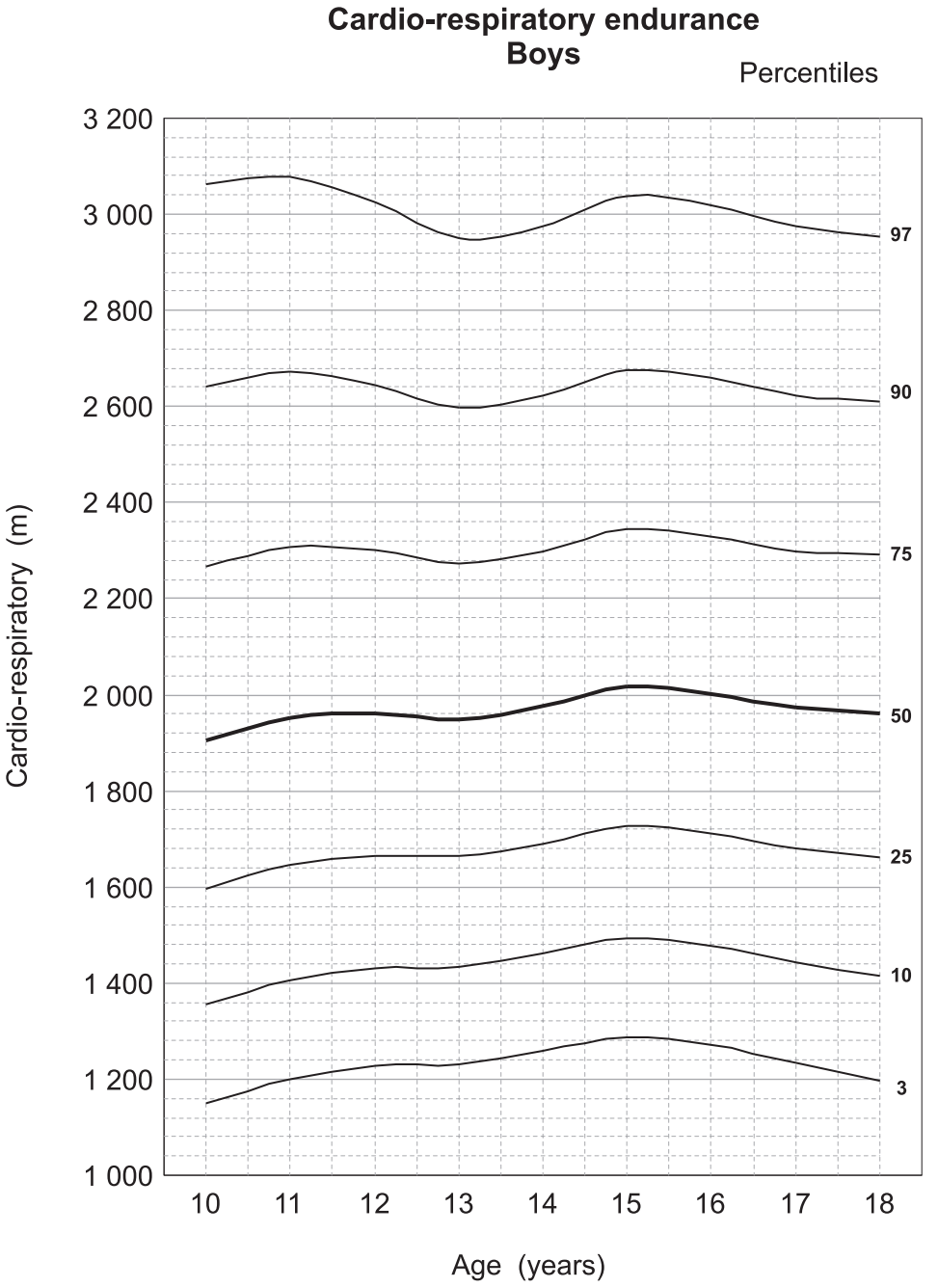



Fig. 14. Centile charts for evaluation of cardio-respiratory endurance in boys by the Cooper Test: 12 minutes running

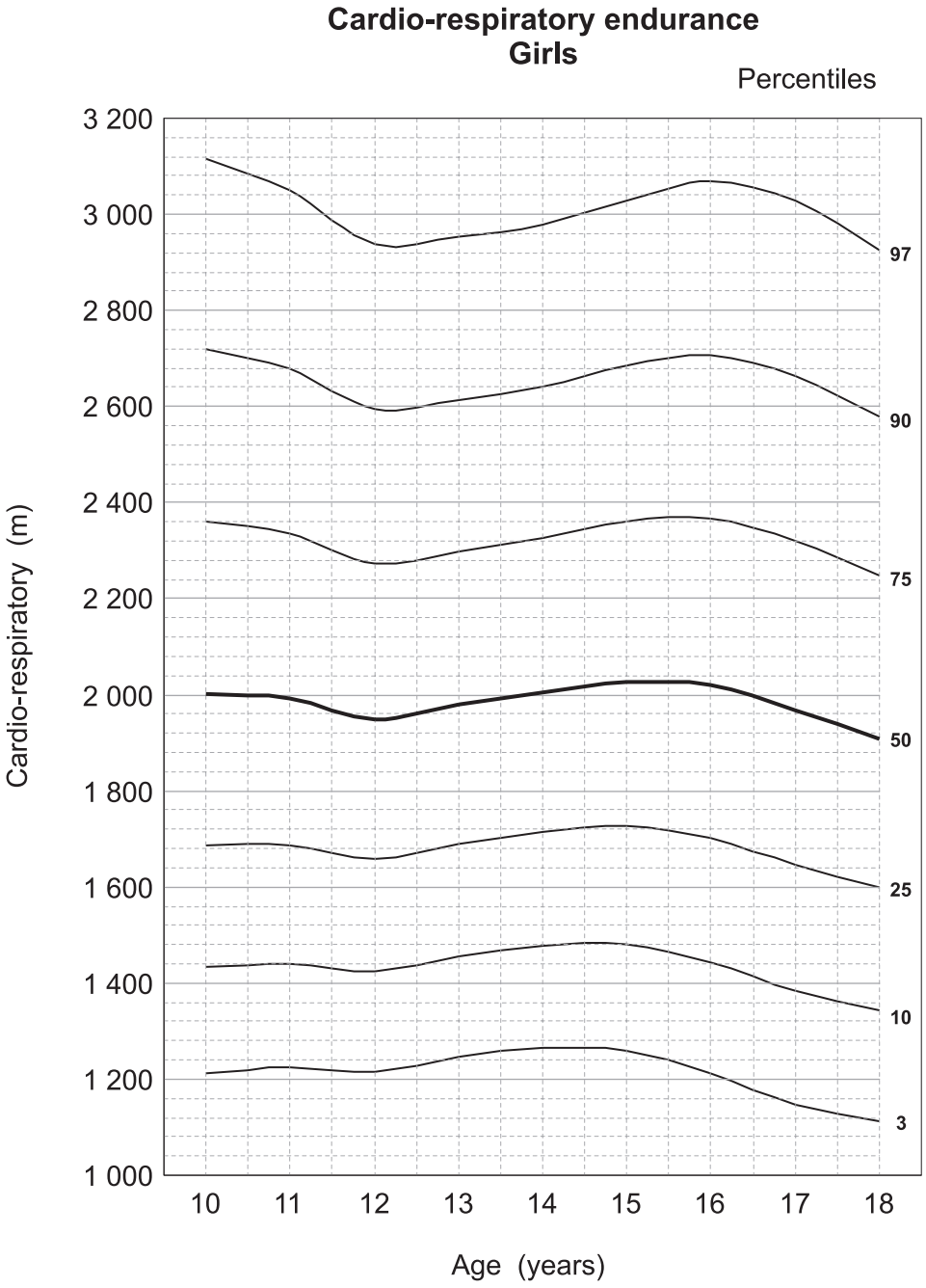



Fig. 14. Centile charts for evaluation of cardio-respiratory endurance in girls by the Cooper Test: 12 minutes running

Table 3. Proportion (%) of examined boys and girls from the region of Wielkopolska in relation to flexibility levels as set out by national standards

| Age | n | Flexibility (sit and reach) | | | | |
|---|---|---|---|---|---|---|
| | | low | below medium | medium | above medium | high |
| | | | Boys | | | |
| 10 | 126 | 25.4 | 15.9 | 48.2 | 8.8 | 1.7 |
| 11 | 274 | 27.4 | 12.8 | 50.0 | 6.9 | 2.9 |
| 12 | 263 | 30.4 | 14.1 | 44.0 | 7.6 | 3.9 |
| 13 | 252 | 25.7 | 12.0 | 48.5 | 10.4 | 3.4 |
| 14 | 252 | 21.0 | 9.5 | 55.2 | 8.7 | 5.6 |
| 15 | 286 | 18.6 | 11.7 | 50.5 | 12.4 | 6.8 |
| 16 | 251 | 23.1 | 15.6 | 49.4 | 5.3 | 6.6 |
| 17 | 253 | 38.4 | 12.0 | 37.6 | 6.6 | 5.4 |
| 18 | 292 | 39.3 | 16.1 | 36.6 | 4.8 | 3.2 |
| | | | Girls | | | |
| 10 | 116 | 16.4 | 13.0 | 45.5 | 19.0 | 6.1 |
| 11 | 273 | 16.5 | 14.3 | 54.8 | 10.3 | 4.1 |
| 12 | 301 | 18.0 | 14.6 | 53.7 | 8.3 | 5.4 |
| 13 | 271 | 16.7 | 15.3 | 52.2 | 10.4 | 5.4 |
| 14 | 269 | 12.7 | 9.3 | 56.0 | 11.6 | 10.4 |
| 15 | 266 | 17.4 | 13.7 | 51.9 | 11.9 | 5.1 |
| 16 | 304 | 21.4 | 16.2 | 46.2 | 11.4 | 4.8 |
| 17 | 184 | 30.1 | 10.1 | 46.0 | 9.0 | 4.8 |
| 18 | 269 | 34.6 | 10.6 | 41.2 | 8.8 | 4.8 |

Table 4. Proportion (%) of examined boys and girls from the region of Wielkopolska in relation to explosive power levels as set out by national standards

| Age | n | Explosive power (standing broad jump) | | | | |
|---|---|---|---|---|---|---|
| | | low | below medium | medium | above medium | high |
| | | | Boys | | | |
| 10 | 125 | 24.7 | 20.8 | 47.6 | 5.0 | 1.9 |
| 11 | 275 | 19.3 | 13.8 | 50.4 | 8.4 | 8.1 |
| 12 | 264 | 17.0 | 17.4 | 49.6 | 8.7 | 7.3 |
| 13 | 253 | 17.8 | 17.0 | 51.5 | 7.6 | 6.1 |
| 14 | 252 | 36.1 | 15.1 | 38.9 | 4.0 | 5.9 |
| 15 | 282 | 37.3 | 13.0 | 34.0 | 8.8 | 6.9 |
| 16 | 247 | 46.2 | 15.9 | 27.3 | 7.2 | 3.4 |
| 17 | 247 | 49.0 | 8.9 | 31.5 | 6.6 | 4.0 |
| 18 | 285 | 53.1 | 10.1 | 25.4 | 5.3 | 6.1 |
| | | | Girls | | | |
| 10 | 116 | 8.7 | 7.0 | 37.8 | 20.7 | 25.8 |
| 11 | 273 | 14.3 | 11.0 | 46.4 | 14.0 | 14.3 |
| 12 | 301 | 12.0 | 8.7 | 49.4 | 12.0 | 17.9 |
| 13 | 266 | 14.8 | 12.4 | 39.8 | 12.0 | 21.0 |
| 14 | 268 | 11.9 | 10.8 | 43.7 | 13.1 | 20.5 |
| 15 | 267 | 9.3 | 11.9 | 33.3 | 11.1 | 34.4 |
| 16 | 307 | 13.5 | 8.9 | 37.6 | 10.8 | 29.2 |
| 17 | 189 | 7.9 | 5.3 | 27.5 | 12.2 | 47.1 |
| 18 | 275 | 9.8 | 6.2 | 35.6 | 11.3 | 37.1 |

Table 5. Proportion (%) of examined boys and girls from the region of Wielkopolska in relation to static strength levels as set out by national standards

| Age | n | Static strength (hand grip) | | | | |
|---|---|---|---|---|---|---|
| | | low | below medium | medium | above medium | high |
| | | | Boys | | | |
| 10 | 127 | – | – | 37.8 | 22.8 | 39.4 |
| 11 | 273 | 4.9 | 3.1 | 47.3 | 15.4 | 29.3 |
| 12 | 263 | 3.5 | 5.4 | 61.4 | 14.5 | 15.2 |
| 13 | 253 | 9.6 | 7.6 | 64.5 | 8.4 | 9.9 |
| 14 | 252 | 7.9 | 16.3 | 59.9 | 6.0 | 9.9 |
| 15 | 284 | 14.3 | 22.2 | 44.2 | 11.5 | 7.8 |
| 16 | 248 | 28.8 | 17.0 | 34.7 | 7.5 | 12.0 |
| 17 | 248 | 26.4 | 13.9 | 33.0 | 12.8 | 13.9 |
| 18 | 288 | 34.1 | 11.6 | 34.1 | 7.8 | 12.4 |
| | | | Girls | | | |
| 10 | 117 | – | 0.8 | 23.9 | 19.7 | 55.6 |
| 11 | 273 | 0.4 | 1.9 | 26.7 | 27.8 | 43.2 |
| 12 | 301 | 1.4 | 1.0 | 47.1 | 16.3 | 34.2 |
| 13 | 272 | – | 2.1 | 44.0 | 14.4 | 39.5 |
| 14 | 269 | 0.8 | 1.9 | 36.0 | 16.4 | 44.9 |
| 15 | 269 | 3.0 | 4.1 | 34.6 | 6.6 | 51.7 |
| 16 | 307 | 2.8 | 5.0 | 26.7 | 12.1 | 53.4 |
| 17 | 189 | 0.5 | 2.7 | 22.2 | 5.8 | 68.8 |
| 18 | 275 | 1.1 | 2.1 | 32.0 | 8.4 | 56.4 |

Table 6. Proportion (%) of examined boys and girls from the region of Wielkopolska in relation to trunk strength levels as set out by national standards

| Age | n | Trunk strength (sit-ups) | | | | |
|---|---|---|---|---|---|---|
| | | low | below medium | medium | above medium | high |
| | | | Boys | | | |
| 10 | 125 | 20,8 | 9,7 | 52,3 | 13,7 | 3,5 |
| 11 | 275 | 9,5 | 13,8 | 53,0 | 14,6 | 9,1 |
| 12 | 264 | 5,3 | 15,1 | 58,0 | 13,6 | 8,0 |
| 13 | 253 | 7,6 | 15,1 | 53,9 | 15,8 | 7,6 |
| 14 | 252 | 7,9 | 8,7 | 61,9 | 10,3 | 11,2 |
| 15 | 282 | 14,0 | 8,2 | 51,1 | 14,7 | 12,0 |
| 16 | 248 | 16,3 | 13,1 | 51,2 | 9,6 | 9,8 |
| 17 | 247 | 13,6 | 10,1 | 54,9 | 12,8 | 8,6 |
| 18 | 284 | 17,7 | 9,5 | 47,7 | 11,5 | 13,6 |
| | | | Girls | | | |
| 10 | 116 | 1,0 | 3,6 | 57,5 | 19,0 | 18,9 |
| 11 | 273 | 8,1 | 8,1 | 55,9 | 8,8 | 19,1 |
| 12 | 301 | 8,0 | 5,3 | 48,4 | 16,3 | 22,0 |
| 13 | 269 | 12,5 | 5,6 | 46,5 | 10,6 | 24,8 |
| 14 | 269 | 6,4 | 5,6 | 51,5 | 17,9 | 18,6 |
| 15 | 269 | 5,5 | 5,9 | 39,3 | 17,9 | 31,4 |
| 16 | 306 | 4,1 | 1,9 | 44,4 | 18,0 | 31,6 |
| 17 | 189 | 2,1 | 2,7 | 31,2 | 18,5 | 45,5 |
| 18 | 275 | 2,9 | 0,7 | 43,7 | 12,7 | 40,0 |

Table 7. Proportion (%) of examined boys and girls from the region of Wielkopolska in relation to functional strength levels as set out by national standards

| Age | n | Functional strength (bent arm hang) | | | | |
|---|---|---|---|---|---|---|
| | | low | below medium | medium | above medium | high |
| | | | Boys | | | |
| 10 | 126 | 48,9 | 15,2 | 34,1 | 0,9 | 0,9 |
| 11 | 274 | 37,8 | 13,9 | 38,9 | 6,6 | 2,8 |
| 12 | 263 | 41,0 | 11,1 | 37,6 | 5,3 | 5,0 |
| 13 | 246 | 39,9 | 8,5 | 42,7 | 5,8 | 3,1 |
| 14 | 252 | 44,0 | 16,7 | 32,5 | 4,8 | 2,0 |
| 15 | 282 | 44,2 | 17,8 | 30,8 | 5,1 | 2,1 |
| 16 | 248 | 59,6 | 9,6 | 25,0 | 4,5 | 1,3 |
| 17 | 245 | 60,0 | 12,2 | 20,3 | 4,8 | 2,7 |
| 18 | 285 | 59,6 | 12,1 | 21,7 | 2,6 | 4,0 |
| | | | Girls | | | |
| 10 | 114 | 24,5 | 11,6 | 45,8 | 7,3 | 10,7 |
| 11 | 270 | 31,7 | 7,6 | 45,2 | 9,4 | 6,1 |
| 12 | 301 | 28,6 | 7,3 | 44,4 | 10,6 | 9,1 |
| 13 | 271 | 30,8 | 6,9 | 45,0 | 11,2 | 6,1 |
| 14 | 269 | 30,9 | 8,2 | 35,6 | 12,3 | 13,0 |
| 15 | 265 | 21,8 | 10,2 | 35,6 | 13,5 | 18,9 |
| 16 | 307 | 17,3 | 9,9 | 37,9 | 14,1 | 20,8 |
| 17 | 189 | 19,6 | 7,9 | 23,8 | 9,5 | 39,2 |
| 18 | 274 | 13,2 | 8,8 | 32,4 | 7,7 | 37,9 |

Table 8. Proportion (%) of examined boys and girls from the region of Wielkopolska in relation to running speed/agility levels as set out by national standards

| Age | n | Running speed/agility (shuttle run: 10×5 m) | | | | |
|---|---|---|---|---|---|---|
| | | low | below medium | medium | above medium | high |
| | | | Boys | | | |
| 10 | 126 | 19.2 | 19.8 | 55.2 | 4.9 | 0.9 |
| 11 | 275 | 11.7 | 12.4 | 58.8 | 11.3 | 5.8 |
| 12 | 264 | 15.9 | 18.2 | 39.0 | 22.0 | 4.9 |
| 13 | 253 | 21.7 | 16.2 | 44.9 | 11.1 | 6.1 |
| 14 | 251 | 17.5 | 20.7 | 51.3 | 8.4 | 2.1 |
| 15 | 283 | 20.8 | 14.6 | 53.8 | 7.2 | 3.6 |
| 16 | 246 | 33.3 | 21.9 | 38.0 | 5.3 | 1.5 |
| 17 | 247 | 30.3 | 20.2 | 44.7 | 2.4 | 2.4 |
| 18 | 281 | 34.6 | 17.6 | 37.0 | 6.6 | 4.2 |
| | | | Girls | | | |
| 10 | 115 | 8.9 | 2.9 | 42.2 | 21.7 | 24.3 |
| 11 | 273 | 23.4 | 4.1 | 49.3 | 11.0 | 12.2 |
| 12 | 300 | 21.0 | 7.1 | 42.5 | 14.4 | 15.0 |
| 13 | 266 | 18.5 | 13.8 | 43.1 | 10.1 | 14.5 |
| 14 | 268 | 12.4 | 10.2 | 43.5 | 14.9 | 19.0 |
| 15 | 266 | 10.5 | 8.7 | 51.2 | 14.4 | 15.2 |
| 16 | 307 | 7.3 | 12.5 | 53.1 | 16.7 | 10.4 |
| 17 | 188 | 4.9 | 8.0 | 39.8 | 25.5 | 21.8 |
| 18 | 275 | 5.1 | 7.3 | 43.6 | 20.0 | 24.0 |

Table 9. Proportion (%) of examined boys and girls from the region of Wielkopolska in relation to cardio-respiratory endurance levels as set out by national standards

| Age | n | Cardio-respiratory endurance (Cooper Test: 12 minutes running) | | | | |
|---|---|---|---|---|---|---|
| | | low | below medium | medium | above medium | high |
| | | | Boys | | | |
| 10 | 125 | 12.2 | 50.0 | 29.5 | 8.3 | – |
| 11 | 275 | 10.6 | 36.6 | 38.8 | 12.4 | 1.6 |
| 12 | 260 | 13.9 | 32.9 | 38.9 | 12.0 | 2.3 |
| 13 | 249 | 18.1 | 32.6 | 32.2 | 15.0 | 2.1 |
| 14 | 237 | 16.7 | 32.9 | 30.2 | 15.0 | 5.2 |
| 15 | 276 | 21.3 | 28.2 | 24.4 | 19.2 | 6.9 |
| 16 | 237 | 34.4 | 28.1 | 22.6 | 12.4 | 2.5 |
| 17 | 245 | 32.0 | 30.9 | 25.0 | 8.7 | 3.4 |
| 18 | 266 | 43.2 | 16.8 | 23.0 | 11.7 | 5.3 |
| | | | Girls | | | |
| 10 | 115 | 6.3 | 17.4 | 32.8 | 32.8 | 10.7 |
| 11 | 271 | 18.1 | 20.3 | 25.0 | 25.4 | 11.2 |
| 12 | 298 | 16.1 | 27.8 | 30.4 | 16.8 | 8.9 |
| 13 | 262 | 14.1 | 29.6 | 32.5 | 16.6 | 7.2 |
| 14 | 259 | 6.4 | 23.0 | 34.9 | 22.0 | 13.7 |
| 15 | 262 | 8.5 | 21.3 | 38.0 | 19.5 | 12.7 |
| 16 | 304 | 7.2 | 16.5 | 30.7 | 22.3 | 23.3 |
| 17 | 187 | 7.6 | 7.9 | 25.2 | 16.2 | 43.1 |
| 18 | 275 | 4.0 | 12.7 | 32.7 | 20.7 | 29.9 |

cal fitness elements where girls from the Wielkopolska region did worse than the all-Poland population. In all other fitness tests the girls from Wielkopolska achieved higher results. This distinctively marked tendency was visible in the tests for explosive power, static strength and trunk strength. High level of explosive power (Table 4) of girls was demonstrated for the range from 14.3% to 47.1% (at the age of 17). Static strength (Table 5) of the highest level concerned from 34.2% to even 68.8% of girls, also aged 17. In this age category, a low level of static strength concerned maximally 3.0% of girls. In the field of trunk strength (Table 6), there were from 18.6% to 45.5% girls in the highest category (again most of them at the age of 17).

As far as functional strength (upper body endurance), running speed/agility and cardio-respiratory endurance were concerned, a higher proportion of younger girls achieved a similar or lower result in comparison with their peers from the all-Poland research. However, in the higher age categories of girls from Wielkopolska this level is significantly higher. Especially girls aged 17 and 18 years achieved favourable results in the above mentioned physical fitness categories. There were 39.2% and 37.9% of girls respectively that demonstrated a high level of functional strength (Table 7), 21.8% and 24.0% of running speed/agility (Table 8) and 43.1% and 29.9% in the test of cardio-respiratory endurance (Table 9).

In the boys' group, however, the lowest results in each age category were observed, not only in the earlier mentioned flexibility, but also in case of explosive power (Table 4) for 17.0% to 53.1% of boys, functional strength (Table 7) for 37.8%

to 60.0%, running speed/agility (Table 8) for 11.7% to 34.6% and cardio-respiratory endurance (Table 9) for 10.6% to 43.2%. In the above tests the highest proportion of boys taking part in the research with a low physical fitness level occurred in the age categories of 16–18 years. By contrast, high physical fitness level of boys in these tests was demonstrated by respectively 1.9–8.1% (explosive power), 0.9–5.0% (functional strength), 0.9–6.1% (running speed/agility) and 0.0–6.9% in cardio-respiratory endurance.

In the component of trunk strength (Table 6), the studied boys achieved similar results to their peers. Slightly higher results were demonstrated by younger boys in the static strength (Table 5) test (high level for 39.4% at the age of 10 and 29.3% at the age of 11), but the results in this test decline significantly for older boys (low level for 28.8%, 26.4% and 34.1% of boys 16-, 17- and 18-year-old respectively).

The explanation of the reasons for the observed tendencies in the picture of the physical fitness development of children and adolescents from the Wielkopolska region requires additional research on the collected results. Even the most scrupulous analysis of the current view of physical fitness does not entitle to a reliable interpretation of the casual mechanism that condition them. The results are nevertheless surprising and rather far from previous expectations. The physical fitness level of girls is particularly surprising, especially of the oldest ones (17–18 years of age), who performed very well in comparison with the all-Poland's norms. Are these the previously expressed demands of the necessity to focus on the physical education of older girls [Jezierski, 1999] that have finally been met and the result can be observed now? Or maybe it is the development and maturity acceleration has favoured the achievement of better results? Adding the results of the somatic and maturity level characteristics to the analysis can already shed some light on this interpretation.

## Conclusion

1. In certain elements of physical fitness, the children and adolescents from the Wielkopolska region aged 10 to 18 years, boys in particular, demonstrated a significantly lower level in comparison to Poland population in general. In the physical fitness assessment of children and adolescents it has been assumed that the lowest level corresponds to approximately 11.5% of people from the all-Poland population. In the Wielkopolska region it concerned in case of body flexibility from 18.6% to 39.3% and in functional strength from 37.8 to 60.0% of boys in individual age categories and from 12.7% to 34.6% of girls respectively. The physical fitness level of older boys is particularly alarming (16–18 years of age). In the category of explosive power, very low level was demonstrated by 46.2–53.1% of the subjects, in static strength by 28.8–34.1%, in functional strength by 59.6–60.0%, in running speed/agility by 33.3–34.6% and in cardio-respiratory endurance by 34.4–43.2%.

2. In the face of the significantly unfavourable physical fitness levels of boys from the Wielkopolska region versus Poland's population, parents, teachers and class tutors should be addressed to put extra focus on the physical fitness development of boys. It is also necessary, however, to conduct in-depth analysis of the biological, cultural and educational factors which cause this alarming trend.
3. The physical fitness level of girls, apart from flexibility and functional strength, can be regarded as satisfactory. Especially older girls (17–18 year of age) have come out very well in comparison with their peers from the all-Poland tests. The highest range of physical fitness, which also corresponded to approximately 11.5% of the all-Poland population, was demonstrated by 47.1% and 37.1% of girls respectively for explosive power, 68.8% and 56.4% for static strength, 45.5% and 40.0% for trunk strength, 39.2% and 37.9% for functional strength, 21.8% and 24.0% for running speed/agility, and 43.1% and 29.9% for cardio-respiratory endurance. It is crucial to implement supportive measures also in this case, in order to at least maintain the current level of physical fitness.
4. The conducted assessment of physical fitness in population categories is only an introduction to an evaluation of the individual needs of boys and girls that should be analyzed in reference to somatic development level and actual health criteria. This is vital due to many cases of exceptionally low level of physical fitness that have been observed both among boys and girls.

## References

Bouchard C., Shephard R.J.: Physical activity, fitness and health: The model and key concepts. In: C. Bouchard, R.J. Shephard, T. Stephens (Eds.) *Physical Activity, Fitness and Health.* 1994: 77–88. Champain: Human Kinetics Publishers.

Cooper K.H.: *The Aerobics Way*. 1978. London: Corgi Books.

Dobosz J.: Tabele stupunktowe sprawności fizycznej dzieci i młodzieży w Polsce wg testu Eurofit. Maszynopis oraz http://www.kondycjafizyczna.pl/pobierz-materialy.php (20.11.2010)

Docherty D.: Field tests and test batteries. In: D. Docherty (Ed.) *Measurement in Pediatric Exercise Science*. 1996. Champain: Human Kinetics Publishers.

EUROFIT, *European Tests of Physical Fitness*. Second edition. 1993. Strasbourg: Council of Europe.

Franks B.D.: *YMCA Youth Fitness Manual*. 1989. Human Kinetics Publishers.

Jezierski R.: Wychowanie fizyczne dziewcząt w świetle współczesnych oczekiwań i potrzeb. In: W. Osiński, J. Pośpiech, A. Szecowka (Ed.) *Wychowanie fizyczne w dobie reformy edukacji. Oczekiwania – realia – perspektywy.* 1999. Wrocław: Wydawnictwo ATLA-2.

Oja P., Tuxworth B. (Eds): Eurofit for Adults. Assessment of Health-Related Fitness. 1995. Strasbourg: Council of Europe.

Pilicz S., Przewęda R., Dobosz J., Nowacka-Dobosz S.: Physical fitness score tables of Polish youth. Criteria for measuring aerobic capacity by the Cooper Test. 2002. Studia i monografie nr 86. Warszawa: Akademia Wychowania Fizycznego.

Przewęda R., Dobosz J.: Kondycja fizyczna polskiej młodzieży. 2003. Studia i monografie nr 98. Warszawa: Akademia Wychowania Fizycznego.

Skinner J.S., Oja P.: Laboratory and field tests for assessing health-related fitness. In: C. Bouchard, R.J. Shephard, T. Stephens (Eds.) *Physical Activity, Fitness and Health*. 1994: 160–179. Champain: Human Kinetics Publishers.

Stupnicki R., Przewęda R., Milde K.: *Percentile reference curves for physical fitness measured by Eurofit tests in Polish youths*. 2003. Studia i monografie nr 91. Warszawa: Akademia Wychowania Fizycznego.

Wuest D.A, Bucher C.A.: *Foundations of Physical Education and Sport* (11th Ed.). 1996. St. Louis: Mosby Year Book Inc.

Alicja Krzyżaniak, Barbara Stawińska-Witoszyńska,
Małgorzata Krzywińska-Wiewiorowska,
Maria Kaczmarek, Aldona Siwińska

# The distribution of arterial blood pressure in an adolescent population

**Abstract:** The aim of this study was to assess the arterial blood pressure values in children and adolescents in the Wielkopolska province. The study was performed on a representative, randomly selected group of students, aged 10–18, participating in the ADOPOLNOR project. Before taking their blood pressure, each pupil was measured for their arm width (to select a right size of an arm band) and for height and weight. The BP measurements were made before noon, three times at 2-, 3-day intervals in strict compliance with the method presented in Report Four (each measurement was made twice a day). The qualification into the hypertension or normal high pressure (pre-hypertension) groups were based on guidelines of the Working Group on High Blood Pressure in Children and Adolescents. BP values in the studied population were presented in the form of percentile grids based on the Cole's method.

**Key words:** blood pressure, adolescents, reference percentiles

## Introduction

While cardiovascular diseases in adolescents do not represent a significant health problem, bad habits and improper lifestyle, in particular insufficient physical activity, may increase the risk of their occurrence. One of the most dangerous risk factors is elevated blood pressure. Recent studies on young population in Poland indicate that the frequency of arterial hypertension is not at all low [Wobasz 2004]. Epidemiological studies in recent decades have shown that the proportion of adolescents affected with primary hypertension ranges from several to several dozen percent [Krzyżania et al. 2003]. The measurement of blood pressure is therefore becoming ever more relevant. Blood pressure is one of the positive health indicators, but opposite to morphological indicators, it is characterised by a high variability dependent on a number of genetic, prenatal and environmental factors [Kułaga et al. 2009; Neuhauser, Thamm 2007; Nowakowska et al. 2005; Paulus et al. 1999;

Sinaiko et al. 1989; Wyszyńska et al. 1985]. In children and adolescents, the variability may be related to a phase of development. The period of adolescence is of vital importance for the level of blood pressure. It is not only when the size of the left ventricle grows, but also the time when the circumference of the aorta and the lumen of blood vessels increase. Higher blood pressure in adolescents is correlated with the growing weight of left ventricle, which, as recent research shows, is a significant indicator of the risk of ischemic heart disease (IHD). The increase in BP levels is a consequence of hormonal and cardiovascular changes occurring in that period. Children and adolescents entering adulthood with higher BP parameters are more likely to be affected with hypertension in their adult life. Therefore, identification of children with high pressure should be constant component of paediatric preventive care [Oblacinska, Woynarowska 2002]. However, in order to identify cardiovascular risk factors, particularly high blood pressure, representative and updated standards have to be put in place.

Updated blood pressure standards for children and adolescents is essential not only for physicians, paediatricians and primary health care, but also for epidemiologists.

The aim of this paper is to assess the arterial blood pressure values in children and adolescents in the Wielkopolska province.

## Materials and methods

The study was performed on a representative, randomly selected group of students, aged 10–18, participating in the ADOPOLNOR project. Before taking their blood pressure, each pupil was measured for their arm width (to select a right size of an arm band) and for height and weight [RSTFBPCChildren 1987]. The BP measurements were made before noon, three times at 2-, 3-day intervals in strict compliance with the method presented in Report Four (each measurement was made twice a day) [The Fourth Report 2004]. The average of the three measurements taken on three different days constituted the basis for evaluating the prevalence of arterial hypertension. The proportions of pupils with hypertension or high blood pressure were calculated in line with the applicable Polish standard [Krzyżaniak et al. 2003]. The qualification into the hypertension or high normal blood pressure (pre-hypertension) groups were based on guidelines of the Working Group on High Blood Pressure in Children and Adolescents [The Fourth Report 2004]. Pupils whose mean systolic and/or diastolic pressure (at three measurements taken on different days) was ≥ 95 centile, were classified into the hypertension group, while those BP levels at <95 centile, but ≥90 centile were categorised as having high normal blood pressure [The Fourth Report 2004]. In biological terms, the measurement results were benchmarked against the Polish standard that correlates arterial blood pressure with body height [Krzyżaniak et al. 20032]. BP values in the studied population were presented in the form of percentile grids based on the Cole's method [Cole 1992].

## Results

Mean values of systolic blood pressure, both in boys and girls, grew bigger with age. In boys, from 107.4 mmHg at the age of 10 to 121.8 mmHg at the age of 18 (Table 1). In girls, from 106.9 mmHg at the age of 10 to 112.0 mmHg at the age of 18 (Table 2). Diastolic pressure, like systolic one, increased with age from 64.5 mmHg in boys aged 10 to 73.4 mmHg in boys aged 18. In girls, the values were 64.0 and 69.2, respectively. Mean systolic BP levels (broken down by body height, age, gender and measured at three different occasions), which were ≥ 95 centile, thus substantiating further arterial hypertension diagnostics, occurred in 9% of both boys and girls (see Table 3, 4). The prevalence of hypertension and high normal pressure was the highest in boys aged 16, 17 and 18. While the highest proportions of hypertension in girls was found in the 17, 15 and 13 age groups.

High normal pressure, which often requires further observation, was found in 5.5% of boys and 7.1% of girls.

Table 1. Mean values of systolic and diastolic blood pressure in boys ( mmHg)

| Age | N | Systolic blood pressure | | | | Diastolic blood pressure | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | x | SD | min | max | x | SD | min | max |
| 10 | 154 | 107.45 | 8.96 | 88.33 | 133.33 | 64.49 | 6.93 | 46.67 | 83.33 |
| 11 | 276 | 108.79 | 10.21 | 7633 | 150.00 | 65.81 | 7.85 | 48.00 | 93.33 |
| 12 | 255 | 112.38 | 10.45 | 84.67 | 150.00 | 67.13 | 6.69 | 47.67 | 86.67 |
| 13 | 243 | 111.10 | 10.21 | 91.67 | 160.00 | 66.13 | 7.76 | 42.67 | 101.67 |
| 14 | 281 | 115.28 | 10.16 | 90.00 | 148.00 | 66.65 | 7.46 | 43.33 | 88.33 |
| 15 | 279 | 117.18 | 10.34 | 86.67 | 156.67 | 68.10 | 7.68 | 45.67 | 90.00 |
| 16 | 281 | 119.14 | 12.11 | 90.00 | 166.67 | 69.98 | 8.55 | 46.67 | 93.33 |
| 17 | 305 | 120.42 | 10.67 | 91.67 | 163.33 | 72.00 | 7.31 | 54.00 | 97.33 |
| 18 | 357 | 121.78 | 11.79 | 91.67 | 180.00 | 73.41 | 7.84 | 53.33 | 100.00 |

Table 2. Mean values of systolic and diastolic blood pressure in girls ( mmHg)

| Age | N | Systolic blood pressure | | | | Diastolic blood pressure | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | x | SD | min | max | x | SD | min | max |
| 10 | 153 | 106.89 | 9.19 | 85.00 | 146.67 | 63.97 | 6.67 | 50.00 | 81.67 |
| 11 | 276 | 108.07 | 8.79 | 88.33 | 143.33 | 65.09 | 6.97 | 43.33 | 88.33 |
| 12 | 294 | 110.26 | 9.51 | 91.00 | 150.00 | 65.75 | 7.46 | 46.67 | 90.00 |
| 13 | 294 | 112.39 | 10.53 | 91.67 | 146.67 | 66.80 | 7.54 | 43.33 | 96.67 |
| 14 | 259 | 112.72 | 11.28 | 88.33 | 156.67 | 66.49 | 7.54 | 50.00 | 91.67 |
| 15 | 312 | 112.78 | 11.90 | 81.67 | 181.67 | 67.08 | 7.99 | 50.00 | 98.33 |
| 16 | 332 | 113.48 | 10.53 | 88.33 | 151.67 | 68.32 | 7.34 | 50.00 | 91.67 |
| 17 | 236 | 113.15 | 9.70 | 86.67 | 143.33 | 69.44 | 7.05 | 53.33 | 91.67 |
| 18 | 317 | 112.00 | 9.94 | 83.33 | 146.7 | 69.17 | 7.15 | 51.67 | 92.00 |

Table 3. Prevalence of elevated blood pressure values in boys ( mmHg)

| Age | N | High normal | | Hypertension | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | N | % | SBP | | DBP | | SBP+DBP | | | |
| | | | | N | % | N | % | N | % | N | % |
| 10 | 154 | 7 | 4.55 | 7 | 4.55 | 2 | 1.30 | 2 | 1.30 | 11 | 7.14 |
| 11 | 276 | 16 | 5.80 | 7 | 2.54 | 10 | 3.62 | 9 | 3.26 | 26 | 9.42 |
| 12 | 255 | 18 | 7.06 | 9 | 3.53 | 4 | 1.57 | 7 | 2.75 | 20 | 7.84 |
| 13 | 243 | 9 | 3.70 | 4 | 1.65 | 5 | 2.06 | 5 | 2.06 | 14 | 5.76 |
| 14 | 281 | 13 | 4.63 | 16 | 5.69 | 5 | 1.78 | 3 | 1.07 | 24 | 8.54 |
| 15 | 279 | 15 | 5.38 | 8 | 2.87 | 4 | 1.43 | 7 | 2.51 | 19 | 6.81 |
| 16 | 281 | 22 | 7.83 | 13 | 4.63 | 8 | 2.85 | 11 | 3.91 | 32 | 11.39 |
| 17 | 305 | 11 | 3.61 | 17 | 5.57 | 9 | 2.95 | 8 | 2.62 | 34 | 11.15 |
| 18 | 357 | 24 | 6.72 | 17 | 4.76 | 10 | 2.80 | 12 | 3.36 | 39 | 10.92 |
| Total | 2431 | 135 | 5.55 | 98 | 4.03 | 57 | 2.34 | 64 | 2.63 | 219 | 9.01 |

Table 4. Prevalence of elevated blood pressure values in girls ( mmHg)

| Age | N | High normal | | Hypertension | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | N | % | SBP | | CR | | CS+CR | | | |
| | | | | N | % | N | % | N | % | N | % |
| 10 | 153 | 7 | 4.58 | 10 | 6.54 | 3 | 1.96 | 1 | 0.65 | 14 | 9.15 |
| 11 | 276 | 21 | 7.61 | 11 | 3.99 | 6 | 2.17 | 1 | 0.36 | 18 | 6.52 |
| 12 | 294 | 15 | 5.10 | 10 | 3.40 | 7 | 2.38 | 7 | 2.38 | 24 | 8.16 |
| 13 | 294 | 15 | 5.10 | 19 | 6.46 | 4 | 1.36 | 6 | 2.04 | 29 | 9.86 |
| 14 | 259 | 16 | 6.18 | 13 | 5.02 | 5 | 1.93 | 7 | 2.70 | 25 | 9.65 |
| 15 | 312 | 21 | 6.73 | 20 | 6.41 | 4 | 1.28 | 9 | 2.88 | 33 | 10.58 |
| 16 | 332 | 32 | 9.64 | 18 | 5.42 | 6 | 1.81 | 6 | 1.81 | 30 | 9.04 |
| 17 | 236 | 24 | 10.17 | 14 | 5.93 | 10 | 4.24 | 4 | 1.69 | 28 | 11.86 |
| 18 | 317 | 25 | 7.89 | 6 | 1.89 | 6 | 1.89 | 7 | 2.21 | 19 | 5.99 |
| Total | 2473 | 176 | 7.12 | 121 | 4.89 | 51 | 2.06 | 48 | 1.94 | 220 | 8.90 |

BP standards facilitate understanding the growth rate of age-determined BP, particularly with individual assessments. The systolic and diastolic blood pressure values were presented in the form of percentile grids for boys and girls and are shown in the figures and tables (Fig. 1–4, Tables 5, 6).

However, considering the need to allow for the role of body height and weight in contributing to blood pressure values, a BMI-determined diastolic and systolic BP values for boys and girls was also depicted graphically on a percentile grids (Fig. 5–8).

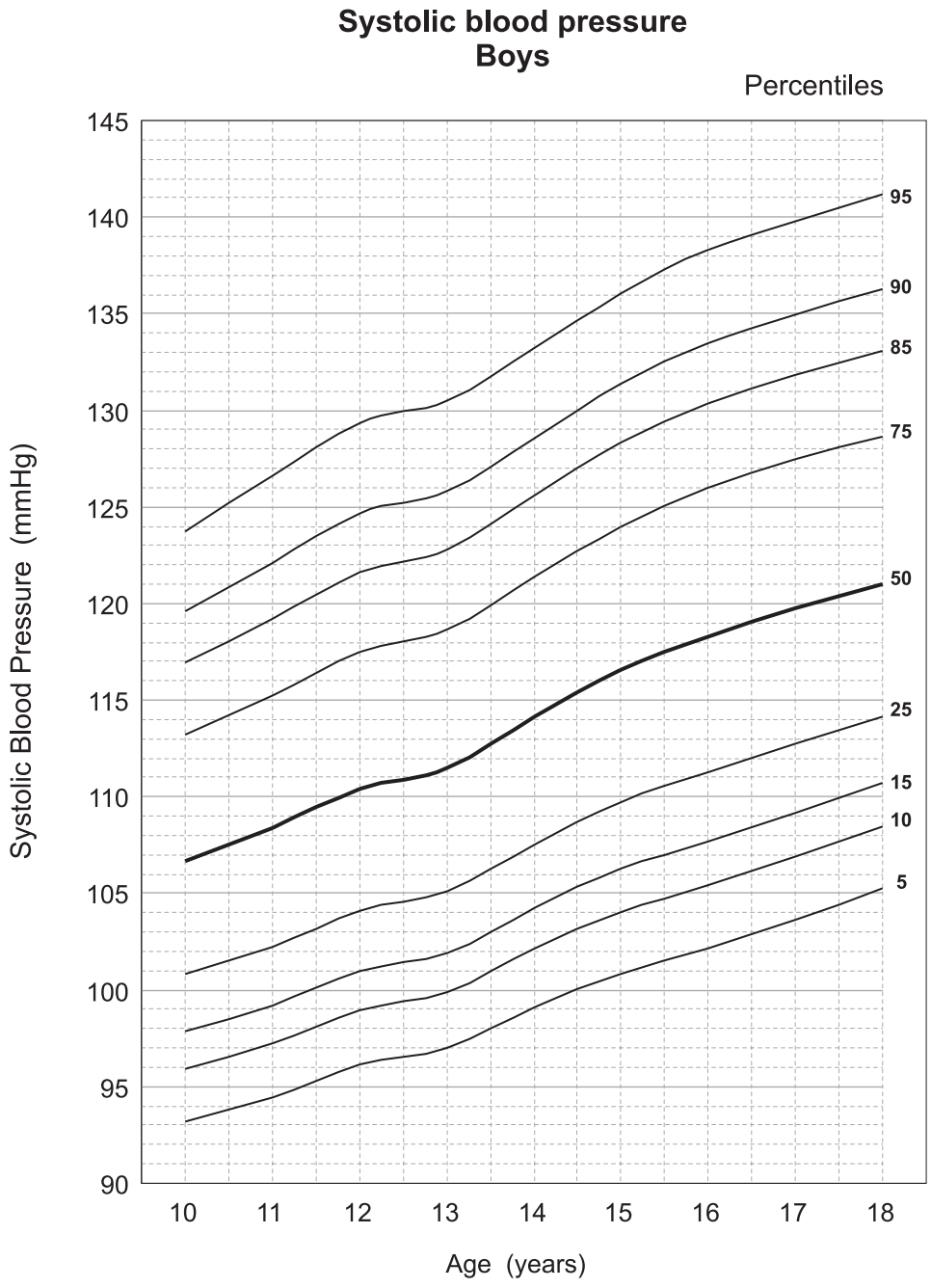



Fig. 1. Systolic blood pressure percentiles in boys

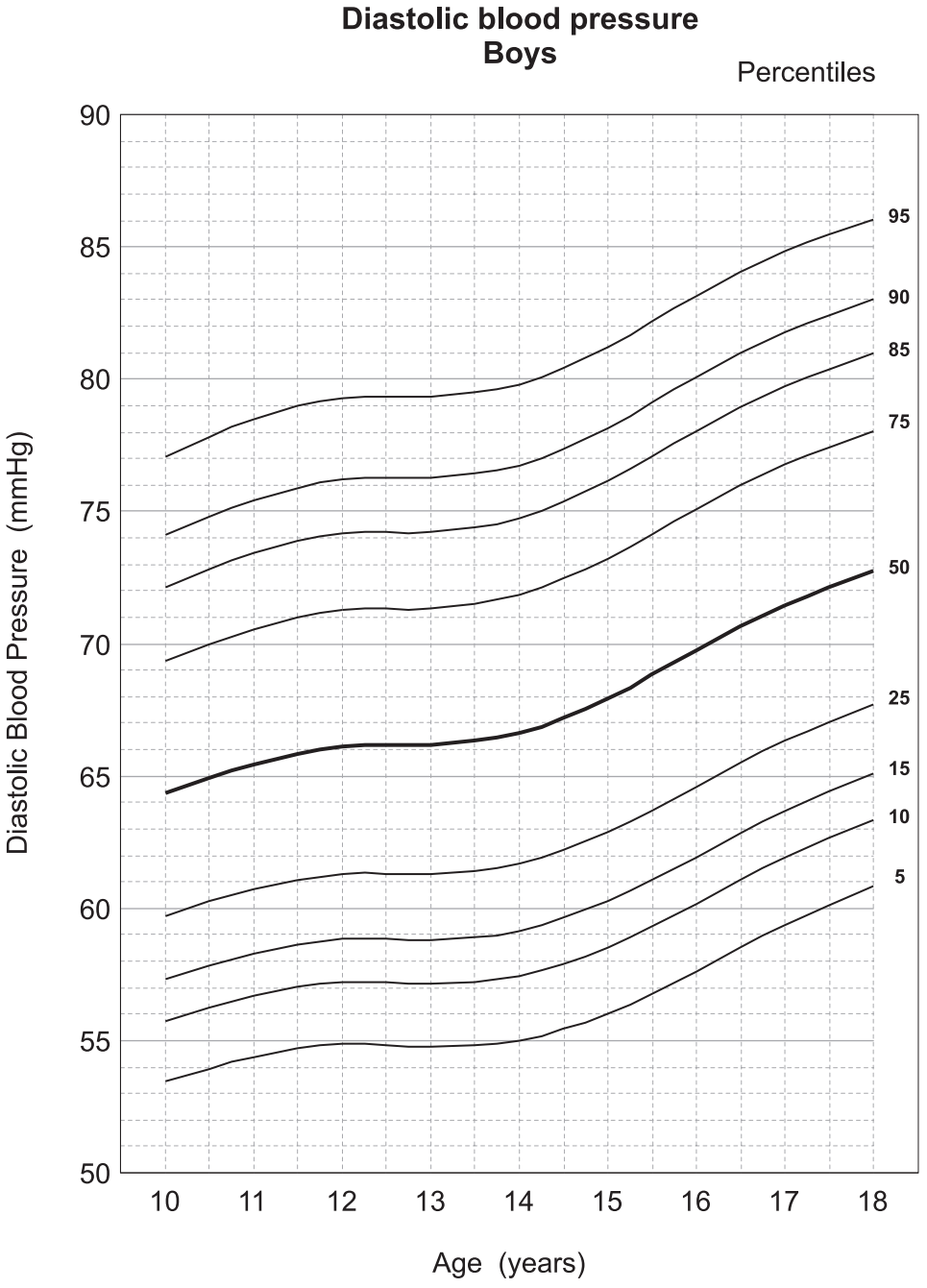



Fig. 2. Diastolic blood pressure percentiles in boys

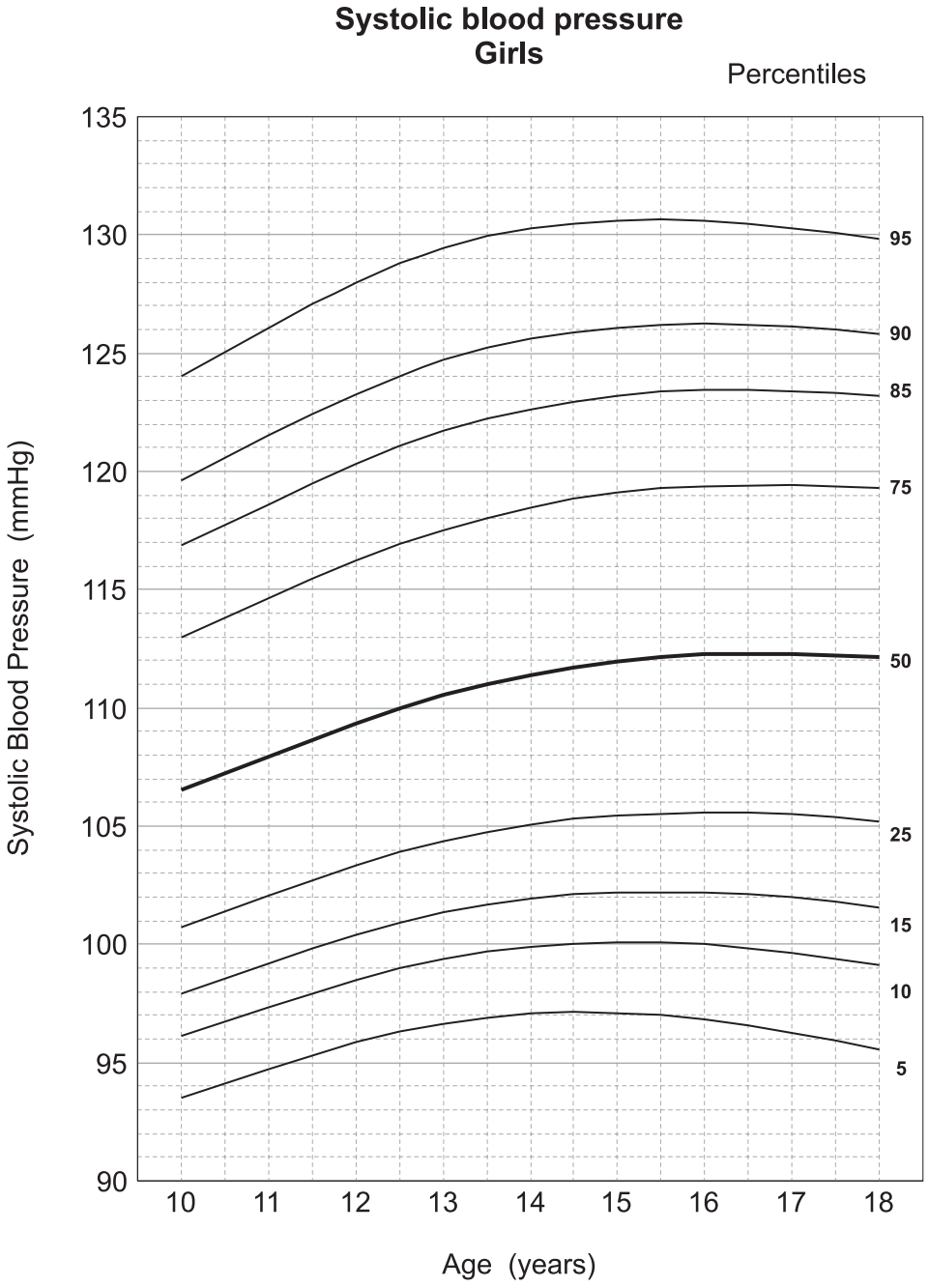



Fig. 3. Systolic blood pressure percentiles in girls

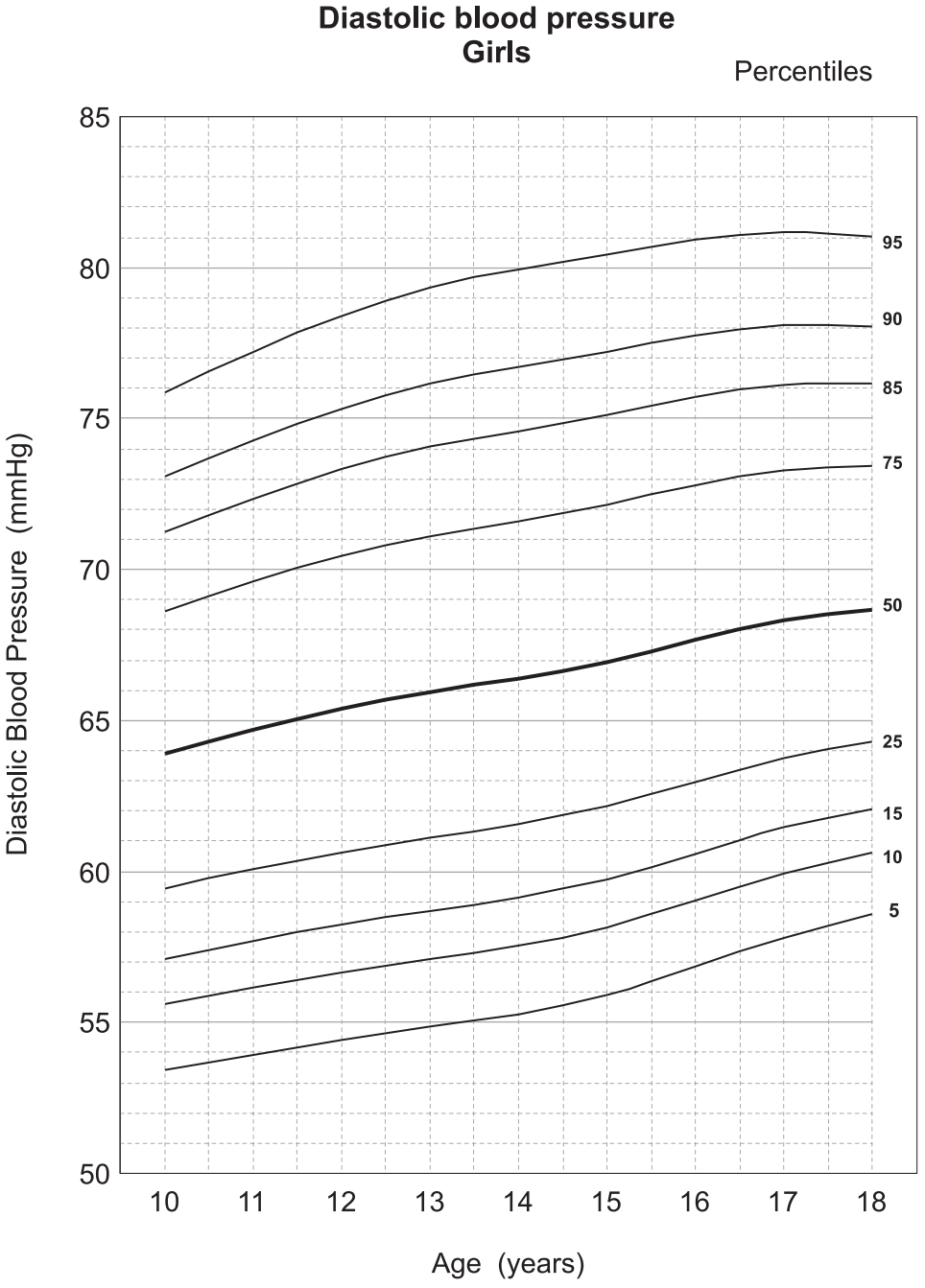



Fig. 4. Diastolic blood pressure percentiles in girls

Table 5. Percentile values of systolic and diastolic blood pressure in boys

| Age | Boys: systolic blood pressure (mmHg) (percentile) | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 3 | 5 | 10 | 15 | 25 | 50 | 75 | 85 | 90 | 95 | 97 |
| 10 | 91.5 | 93.2 | 95.9 | 97.9 | 100.8 | 106.7 | 113.2 | 117.0 | 119.6 | 123.8 | 126.6 |
| 11 | 92.7 | 94.4 | 97.2 | 99.2 | 102.2 | 108.4 | 115.2 | 119.2 | 122.1 | 126.6 | 129.7 |
| 12 | 94.4 | 96.1 | 99.0 | 101.0 | 104.1 | 110.4 | 117.5 | 121.7 | 124.7 | 129.4 | 132.6 |
| 13 | 95.2 | 97.0 | 99.9 | 102.0 | 105.1 | 111.5 | 118.6 | 122.8 | 125.8 | 130.5 | 133.7 |
| 14 | 97.2 | 99.1 | 102.1 | 104.3 | 107.5 | 114.1 | 121.4 | 125.6 | 128.6 | 133.2 | 136.4 |
| 15 | 98.9 | 100.8 | 104.0 | 106.3 | 109.7 | 116.5 | 124.0 | 128.3 | 131.3 | 136.0 | 139.2 |
| 16 | 10.1 | 102.1 | 105.4 | 107.7 | 111.3 | 118.3 | 126.0 | 130.4 | 133.5 | 138.3 | 141.5 |
| 17 | 101.5 | 103.6 | 106.9 | 109.2 | 112.7 | 119.8 | 127.4 | 131.9 | 135.0 | 139.8 | 143.0 |
| 18 | 103.3 | 105.3 | 108.5 | 110.7 | 114.1 | 121.0 | 128.7 | 133.1 | 136.3 | 141.2 | 144.5 |
| Age | Boys: diastolic blood pressure (mmHg) (percentile) | | | | | | | | | | |
| | 3 | 5 | 10 | 15 | 25 | 50 | 75 | 85 | 90 | 95 | 97 |
| 10 | 52.1 | 53.5 | 55.7 | 57.3 | 59.7 | 64.4 | 69.3 | 72.1 | 74.1 | 77.1 | 79.1 |
| 11 | 53.0 | 54.4 | 56.7 | 58.3 | 60.7 | 65.5 | 70.5 | 73.4 | 75.4 | 78.5 | 80.5 |
| 12 | 53.4 | 54.9 | 57.2 | 58.8 | 61.3 | 66.1 | 71.3 | 74.2 | 76.2 | 79.3 | 81.3 |
| 13 | 53.3 | 54.8 | 57.2 | 58.8 | 61.3 | 66.2 | 71.4 | 74.3 | 76.3 | 79.3 | 81.3 |
| 14 | 53.4 | 55.0 | 57.4 | 59.1 | 61.7 | 66.6 | 71.9 | 74.7 | 76.7 | 79.8 | 81.8 |
| 15 | 54.4 | 56.0 | 58.5 | 60.3 | 62.9 | 67.9 | 73.2 | 76.1 | 78.2 | 81.2 | 83.2 |
| 16 | 56.0 | 57.6 | 60.2 | 61.9 | 64.6 | 69.7 | 75.1 | 78.0 | 80.1 | 83.2 | 85.2 |
| 17 | 57.7 | 59.3 | 61.9 | 63.7 | 66.3 | 71.4 | 76.8 | 79.7 | 81.8 | 84.9 | 86.9 |
| 18 | 59.2 | 60.8 | 63.3 | 65.1 | 67.7 | 72.8 | 78.0 | 81.0 | 83.0 | 86.0 | 88.1 |

Table 6. Percentile values of systolic and diastolic blood pressure in girls

| Age | Girls: systolic blood pressure (mmHg) (percentile) | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 3 | 5 | 10 | 15 | 25 | 50 | 75 | 85 | 90 | 95 | 97 |
| 10 | 91.9 | 93.5 | 96.1 | 97.9 | 100.7 | 106.5 | 113.0 | 116.9 | 119.6 | 124.0 | 127.0 |
| 11 | 93.1 | 94.7 | 97.3 | 99.2 | 102.1 | 108.0 | 114.6 | 118.6 | 121.5 | 126.1 | 129.2 |
| 12 | 94.2 | 95.8 | 98.5 | 100.4 | 103.3 | 109.3 | 116.2 | 120.3 | 123.3 | 128.0 | 131.3 |
| 13 | 95.0 | 96.7 | 99.4 | 101.3 | 104.4 | 110.5 | 117.5 | 121.7 | 124.7 | 129.5 | 132.8 |
| 14 | 95.3 | 97.1 | 99.9 | 101.9 | 105.1 | 111.4 | 118.5 | 122.6 | 125.6 | 130.3 | 133.5 |
| 15 | 95.2 | 97.1 | 100.1 | 102.2 | 105.5 | 112.0 | 119.1 | 123.2 | 126.1 | 130.6 | 133.7 |
| 16 | 94.8 | 96.8 | 100.0 | 102.2 | 105.6 | 112.2 | 119.4 | 123.4 | 126.3 | 130.6 | 133.5 |
| 17 | 94.1 | 96.3 | 99.6 | 102.0 | 105.5 | 112.3 | 119.4 | 123.4 | 126.1 | 130.3 | 133.1 |
| 18 | 93.2 | 95.5 | 99.1 | 101.6 | 105.2 | 112.2 | 119.3 | 123.2 | 125.8 | 129.8 | 132.4 |
| Age | Girls: diastolic blood pressure (mmHg) (percentile) | | | | | | | | | | |
| | 3 | 5 | 10 | 15 | 25 | 50 | 75 | 85 | 90 | 95 | 97 |
| 10 | 52.0 | 53.4 | 55.6 | 57.1 | 59.4 | 63.9 | 68.6 | 71.3 | 73.1 | 75.9 | 77.7 |
| 11 | 52.5 | 53.9 | 56.2 | 57.7 | 60.1 | 64.7 | 69.6 | 72.4 | 74.3 | 77.2 | 79.2 |
| 12 | 53.0 | 54.4 | 56.7 | 58.2 | 60.6 | 65.4 | 70.4 | 73.3 | 75.3 | 78.4 | 80.5 |
| 13 | 53.4 | 54.8 | 57.1 | 58.7 | 61.1 | 65.9 | 71.1 | 74.1 | 76.2 | 79.3 | 81.5 |
| 14 | 53.8 | 55.3 | 57.5 | 59.1 | 61.6 | 66.4 | 71.6 | 74.6 | 76.7 | 79.9 | 82.1 |
| 15 | 54.5 | 55.9 | 58.1 | 59.7 | 62.1 | 66.9 | 72.1 | 75.1 | 77.2 | 80.4 | 82.6 |
| 16 | 55.4 | 56.8 | 59.0 | 60.6 | 63.0 | 67.7 | 72.8 | 75.7 | 77.8 | 80.9 | 83.1 |
| 17 | 56.5 | 57.8 | 59.9 | 61.4 | 63.7 | 68.3 | 73.3 | 76.1 | 78.1 | 81.2 | 83.2 |
| 18 | 57.3 | 58.6 | 60.6 | 62.1 | 64.3 | 68.7 | 73.4 | 76.1 | 78.1 | 81.0 | 83.0 |

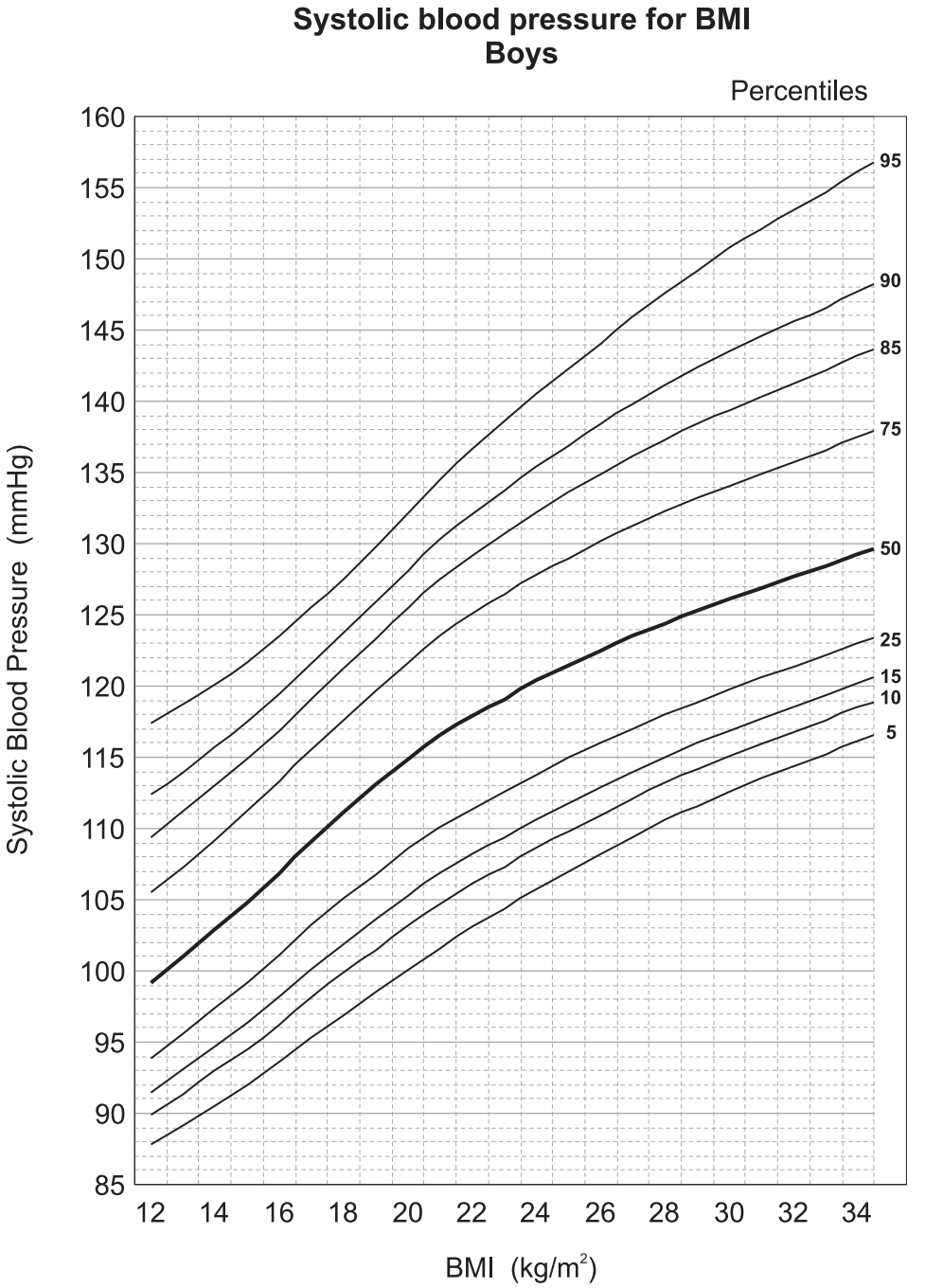



Fig. 5. Systolic blood pressure values in boys in BMI categories

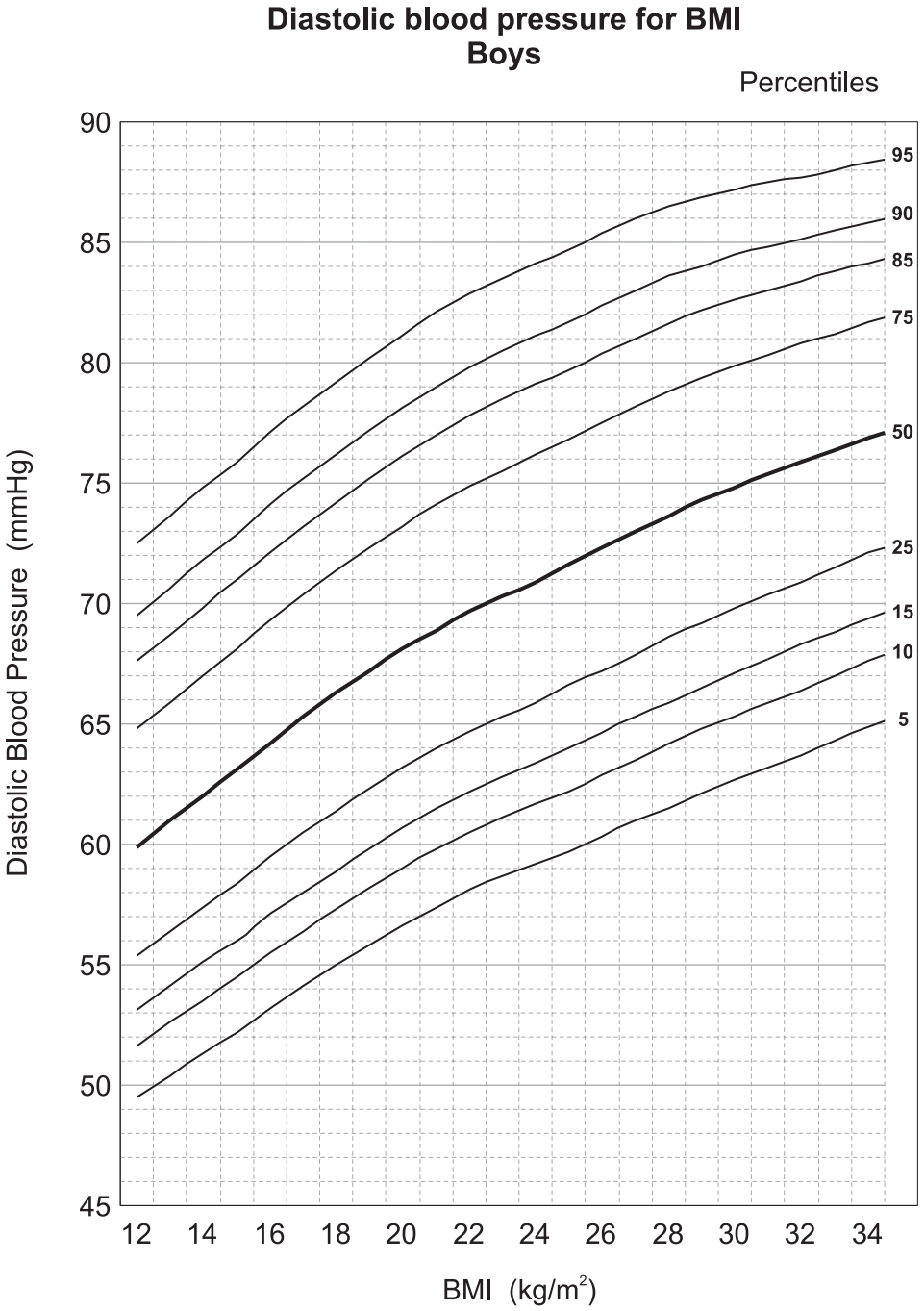



Fig. 6. Diastolic blood pressure values in boys in BMI categories

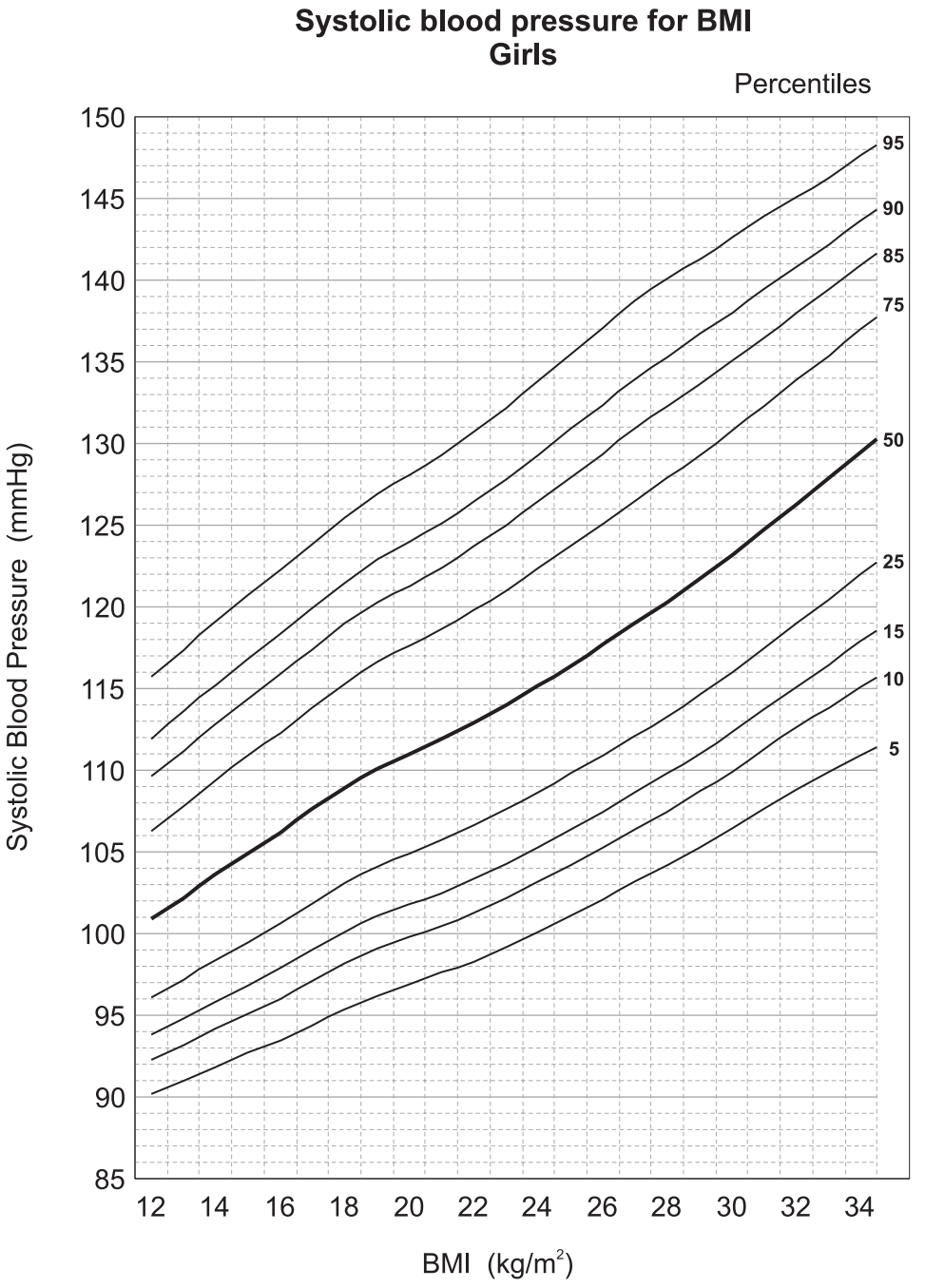



Fig. 7. Systolic blood pressure values in girls in BMI categories

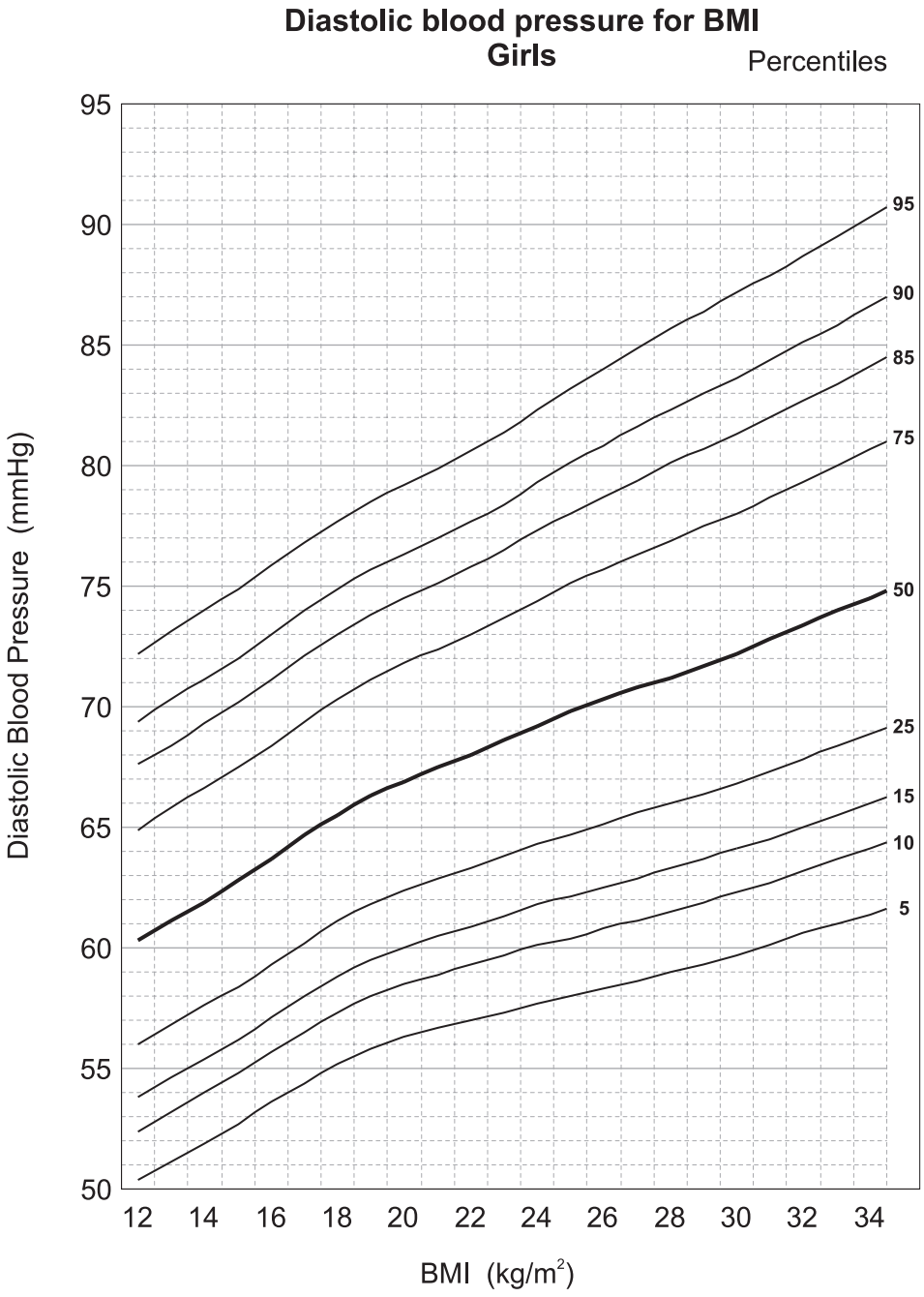



Fig. 8. Diastolic blood pressure values in girls in BMI categories

## Discussion

A particularly important period, which may require a review of blood pressure, is the age of pubescence. While the growth in the BP level in that period may be of a temporary nature, it needs to be borne in mind that higher blood pressure at the time of childhood increases the risk of hypertension at adult age, a phenomenon known as tracking.

The comparison of our study with those of American authors, as presented in the Reports, is possible only for the Report 2, where mean values and standard deviations are given [10]. Current BP levels in Wielkopolska children are significantly higher than those of US children as measured in 1987 [10]. It concerns particularly systolic pressure in boys and girls. As for as diastolic pressure is concerned significantly higher levels were observed in our study (in comparison with studies presented in the 2nd Report) – in boys 15–18 years of age, and in girls 16–18 years of age (Table 7, 8).

BP measurement should be one of principal components of a paediatric examination. It is recommended that a pupil should have his/her blood pressure taken at every visit at a doctor or a nurse. The measurement has to be made in the following cases: kidney, heart, nervous system diseases; hormonal treatment (with steroids, contraceptives, cyclosporin); prior to surgery; qualification to sport activities and professional sport practice; fitness to work examination and pupils' preventive medical examination. Preventive medical examinations of pupils with obligatory BP measurement should be performed at 3rd grade of primary school, first grade of secondary school, and first and last grade of high school [Oblacińska, Woynarowska 2005].

Results of epidemiological studies, both our own and those of other authors, show that the prevalence of hypertension and prehypertension is particularly high with children at their final primary and secondary school grades [Nowakowska

Table 7. Systolic blood pressure norms – comparison between ADPOLNOR and II Report

| Boys | | | | | | | age | Girls | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| ADOPOLNOR | | | II Raport | | | istotność | | ADOPOLNOR | | | II Raport | | | istotność |
| x | N | SD | x | N | SD | | | x | N | SD | x | N | SD | |
| 107.45 | 152 | 8.96 | 101.90 | 1453 | 10.50 | ** | 10 | 106.89 | 152 | 9.19 | 101.8 | 1351 | 10.9 | ** |
| 108.79 | 275 | 10.21 | 103.20 | 1301 | 10.80 | ** | 11 | 108.07 | 275 | 8.79 | 104.6 | 1234 | 11.1 | ** |
| 112.38 | 253 | 10.45 | 105.80 | 1352 | 10.80 | ** | 12 | 110.26 | 295 | 9.51 | 107.5 | 1303 | 11.5 | ** |
| 111.10 | 233 | 10.21 | 107.80 | 4056 | 12.60 | ** | 13 | 112.39 | 283 | 10.53 | 107.2 | 4248 | 12.1 | ** |
| 115.28 | 276 | 10.16 | 110.10 | 3469 | 12.90 | ** | 14 | 112.72 | 253 | 11.28 | 107.8 | 3042 | 11.8 | ** |
| 117.18 | 274 | 10.34 | 113.00 | 3734 | 12.50 | ** | 15 | 112.78 | 309 | 11.90 | 107.5 | 3963 | 11.4 | ** |
| 119.14 | 277 | 12.11 | 114.70 | 2650 | 12.30 | ** | 16 | 113.48 | 331 | 10.53 | 109.1 | 2364 | 11.2 | ** |
| 120.42 | 300 | 10.67 | 117.60 | 3010 | 12.20 | ** | 17 | 113.15 | 234 | 9.70 | 109.9 | 3089 | 11.1 | ** |
| 121.78 | 343 | 11.79 | 118.70 | 1336 | 12.40 | ** | 18 | 112.00 | 315 | 9.94 | 110.0 | 1157 | 10.9 | ** |

Fig. 8. Diastolic blood pressure norms – comparion between ADPOLNOR and II Report

| Boys | | | | | | | age | Girls | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| ADOPOLNOR | | | II Raport | | | istotność | | ADOPOLNOR | | | II Raport | | | istotność |
| x | N | SD | x | N | SD | | | x | N | SD | x | N | SD | |
| 64.49 | 154 | 6.93 | | | | | 10 | 63.97 | 153 | 6.67 | | | | |
| 65.81 | 276 | 7.85 | b.d. dla V tonu | | | | 11 | 65.09 | 276 | 6.97 | b.d. dla V tonu | | | |
| 67.13 | 255 | 6.69 | | | | | 12 | 65.75 | 294 | 7.46 | | | | |
| 66.13 | 233 | 7.76 | 65.5 | 3891 | 10.8 | | 13 | 66.80 | 283 | 7.54 | 67.40 | 4094 | 10.70 | |
| 66.65 | 276 | 7.46 | 66.2 | 3280 | 10.9 | | 14 | 66.49 | 253 | 7.54 | 67.60 | 2861 | 10.60 | |
| 68.10 | 274 | 7.68 | 66.2 | 3555 | 11.0 | ** | 15 | 67.08 | 309 | 7.99 | 66.20 | 3816 | 10.20 | |
| 69.98 | 277 | 8.55 | 67.4 | 2471 | 11.1 | ** | 16 | 68.32 | 331 | 7.34 | 67.00 | 2193 | 10.70 | * |
| 72.00 | 300 | 7.31 | 70.2 | 2827 | 10.4 | ** | 17 | 69.44 | 234 | 7.05 | 67.60 | 2953 | 10.10 | ** |
| 73.41 | 343 | 7.84 | 71.9 | 1179 | 10.1 | * | 18 | 69.17 | 315 | 7.15 | 67.40 | 993 | 10.40 | ** |

et al. 2005; Watkins et al. 2004; Wyszyńska et al. 1985]. This indicates the need for more frequent BP measurements at that period, in particular for pupils at their final year of secondary school, as this may often be their last preventive examination with obligatory BP control. BP standards for children and adolescents have gone through a variety of modifications, as they have for adults; however, the need to take account of age, gender and body height has always been underlined in this respect.

Current guidelines of the US Working Group on High Blood Pressure in Children and Adolescents (Fourth Report) recommend arterial blood pressure to be evaluated against standards presented in tables where BPs are categorised by body weight with consideration given to gender and age. A standard of that type for Poland was published in 2009 [2].

The results presented in the current study in the form of percentile grids will allow to capture the development dynamic, particularly in the period of adolescence, where the growth rate in body height is high.

A disparity of more than two percentile channels between particular features of height and blood pressure may be an indication of developmental irregularities.

## Conclusion

High proportions of pupils with elevated BP levels, giving grounds to a hypertension diagnosis, should encourage more frequent BP measurements in order to take preventive measures to control this crucial health risk factor.

## References

Cole T.J., Grenn P.J.: Smoothing reference centile curves: the LMS method and penalized likehood. *Stat Med*. 1992; 11: 1305–1319.

Krzyżaniak A., Krzywińska-Wiewiorowska M., Stawińska-Witoszyńska B. et al.: Blood pressure references for Polish children and adolescents. *J. of Pediatrics* 2009; 168: 1335–1342.

Krzyżaniak A., Stawińska-Witoszyńska B., Szylagyi-Pągowska I.: Ciśnienie tętnicze dzieci i młodzieży województwa mazowieckiego i wielkopolskiego. *Przegl. Lek*. 2003; 60, 81–85.

Kułaga Z., Litwin M., Zajączkowska M.M. et al.: Regionalne różnice parametrów antropometrycznych oraz ciśnienia tętniczego uczniów w wieku 7–18 lat. *Problemy Higieny i Epidemiologii* 2009; 90(1): 32–41.

Neuhauser H., Thamm M.: Blood pressure measurement In the German Health Interview and Examination Survey for Children and Adolescents (KiGGS). Methodology and initial results. *Bundesgesundheitsblatt Gesundheitsforschung Gesundheitschutz* 2007; 50(5–6): 728–735. Doi: 10.1007/s00103-007-0234-6

Nowakowska D., Zwolińska D., Makulska I.: Nadciśnienie tętnicze u dzieci i młodzieży szkolnej zamieszkałych w Kluczborku. *Wiadomości Lek*. 2005; 8, 29–34.

Oblacińska A., Woynarowska B.: *Profilaktyczne badania lekarskie I inne zadania lekarza w opiece zdrowotnej nad uczniami. Poradnik dla lekarzy opieki zdrowotnej*. Instytut Matki i Dziecka, Zakład Medycyny Szkolnej, Warszawa 2002.

Ogólnopolskie i regionalne rozpowszechnianie głównych czynników ryzyka chorób układu sercowo-naczyniowego w Polsce. Wyniki badania WOBASZ. *Kardiologia Polska* 2004; 61 (Suppl. 4): 1–26.

Paulus D., Saint-Remy A., Jeanjean M.: Blood pressure during adolescence: a study among Belgian adolescents selected from a high cardiovascular risk population. *Eur. J. Epidemiol* 1999, 15(9): 783–790. Doi: 10.1023/A:1007670613848.

Report of the Second Task Force on Blood Pressure Control in Children. *Pediatrics* 1987; 79(1): 1–25.

Sinaiko A.R., Gomez-Marin O., Prineas R.J.: Prevelence of "singnificant" hypertension in junior high school-aged children: the Children and Adolescent Blood Pressure Program. *J. Pediatr*. 1989; 114(1): 664–669. Doi: 10.1016/S0022-3476(89) 80718-8.

The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children Adolescents National High Blood Pressure Education Program Working Group of High Blood pressure in Children Adolescents. *Pediatrics* 2004; 114: 555–576.

Watkins D., McCarron P., Cran G., Boreham et al.: Trends in blood pressure over 10 years in adolescents: analyses of cross sectional surveys in the Northen Ireland Young Hearts project. *BMI* 2004; 29(7458): 139. Doi: 10.1136/bmj.38149519139.7C

Wyszyńska T., Skibicka-Regulska Z., Frelek M., Cichocka E.: Nadciśnienie tętnicze u młodzieży szkolnej – ocena częstości występowania i przyczyn. *Pediatric Pal*. 1985; 2: 169–176.

# Quality of Life

Maria Kaczmarek

# Implications of socio-cultural and lifestyle factors for the quality of life in adolescence

**Abstract:** This chapter is composed of two parts. The first part presents a theoretical background for understanding the concept of quality of life and psychological well-being applied to human auxology. The second part is a report on variation in the generic quality of life (QoL) during the period of adolescence within the social and cultural contexts. The objective of the empirical study was to identify socioeconomic factors and adolescent lifestyle health choices as single predictors of self-perceived quality of life. This was a self-administered cross-sectional survey available by paper-and-pencil and conducted during school time under the ADOPOLNOR project, between May 2009 and June 2010. A YQOL-R model for generic QoL has been administered to 2,758 young people aged 13–18, randomly selected from general population, students of the junior and senior secondary schools. Along with this, two other instruments were used, the ADOPOLNOR-M addressed to adolescents and ADOPOLNOR-R addressed to parents. A series of uni- and multivariate ANOVA/ANCOVA models were conducted under control of chronological age. The findings revealed gender disparities in the QoL with girls more likely to be sensitive to contextual factors than boys. Gender, family economic status, self-rated health and health disability as well as physical activity and cigarette smoking were predictors of QoL. The findings fill the gap in knowledge of quality of life in general population of adolescents. This knowledge, essential to assess how adolescents perceive their own situation, may be used for both further theoretical development and translation to practice aiming at consideration of intervention strategy in the rising tide of child and adolescent physical and mental health.

**Key words:** psychosocial well-being, socioeconomic status, family wealth, lifestyle behaviour, self-rated health, youth disability

*The only questions worth asking today are whether humans are going to have any emotions tomorrow, and what the quality of life will be if the answer is no.*
Lester Bangs (1948–1982)

*Self-esteem is as important to our well-being as legs are to a table. It is essential for physical and mental health and for happiness*
Louise Hart

## Quality of life

Quality of life (QoL) is a concept used in practice by economists, politicians, doctors, sociologists, psychologists, ethicists and anthropologists to evaluate social and individual well-being Langlois and Anderson 2002]. Along with the adoption of a new definition of health proposed by WHO, whereby health is a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity [WHO 1946], the concept of the quality of life has acquired a new meaning as an added value to health. This approach has been reflected by the growth in the number of publications dealing with quality of life in scientific research and clinical practice. In comparison, the PubMed database for 1973 shows only 5 original scientific articles with "quality of life" among their keywords versus 1,252 in 1993 [Testa 1996]. Nowadays, as estimated by Armstrong and Caldwell, 5,000 articles on different aspects of QoL are published every year [Armstrong and Caldwell 2004]. The application of advanced technologies to diagnose and treat certain diseases has turned doctors' attention towards the quality of life rather then merely saving life or lengthening survival.

Over the recent two decades, QoL seen in a life-span perspective has become one of key issues for researchers, international programmes and local organisations, as well as authorities responsible for health and social policy. In 1999–2002, the quality of life was one of the research priorities of the 5th EU Framework Programme and of many programmes and projects inspired and financed by WHO. The programmes were aimed to solve the problem of QoL of ageing individuals and societies and respond to the question of whether the lengthening of human life is not achieved at the expense of its quality, i.e. well-being and happiness. The problems of children and adolescents, particularly those with disabilities, were also addressed, e.g. by the sister projects KIDSCREEN/DISABKIDS carried out under the 5th EU Framework Programme.

At present, one of the biggest challenges faced by Europe's and world's health policy is to level out inequalities in the area of health. Particular care is given to children, adolescents and family. In February 2005, in response to the alarming processes of social stratification and in equality, WHO established the Commission on Social Determinants of Health. The Commission's task was to monitor economic and social living conditions and their impact on the health status and well being of individuals and societies, and to counteract marginalisation of elderly, disabled or socially excluded persons. The concept of the quality of life plays a key role in abovementioned efforts.

Quality of life measurements are used as parameters of social life indicating changes that occur in particular areas of life. In Poland, systematic, wide-scale population-based studies on quality of life have been conducted since 1991 allowing to evaluate the population's level of satisfaction with life [Czapiński and Panek 2011].

The number of publications on various aspects of QoL is truly impressive. They include theoretical papers, proposals of new instruments to measure QoL, results of empirical investigations, review articles, discussions and meta-analyses.

## Short overview of the inception and development of the concept of QoL

The issue of evaluating human quality of life emerged together with the questions concerning human existence and the value of human life. It first came into being as a philosophical reflection to gradually infiltrate other areas of science. Economics and Sociology focused on material living conditions. Sir James Sinclair (1754–1835), a politician, lawyer and reformer of agriculture, was the first to carry out a regular census of population and property in England based on modern statistical methods [Plackett 1986]. For centuries, material status of a population was the indicator of its quality of life. At the beginning of the 1960s, Hadley Cantril developed scales for overall evaluation of a human living environment, laying foundation for the concept of the QoL based on individual cognitive experience. Then, he applied the same tools to investigate aspirations and satisfaction levels of people living in 13 different countries [Cantril 1965]. The concept of life satisfaction was immediately incorporated in studies on subjective experience in specific areas of life, particularly those related to work and marriage. Norman Bradburn [1969] was one of the first researchers to examine emotional aspects of various types of experience. To this end, he developed and utilised the affect balance scale. He was primarily interested in subjective feelings associated with everyday activities.

In the 1950s, attempts were made to evaluate subjective experiences of large populations by using procedures known from psychiatric practice. Among projects of the highest relevance for social policy, worth noting are the study on mental health of the Yorkville community and the investigation conducted in Sterling county, USA. Of historic importance were also the first nation-wide studies on stress, in particular the psychological and emotional aspects of it, carried out under the Joint Commission on Mental Illness and Health [Campbell 1976]. The results proved usefulness of the questionnaire concerning symptoms, experience and general well being in evaluating QoL, as compared to questions contained in the previously used QoL tool. More and more often, statistics on economic well-being of a population started to be supplemented with a number of indicators describing non-material aspects of life, such as feelings, behaviours and individual development.

The 1970s saw a real breakthrough in quality of QoL research. In 1971, a research programme was launched to measure a level of life satisfaction among US population. The author of the programme, August Campbell, with his associates developed a new scale to measure the level of life satisfaction, also referred to as subjective well-being [Campbell and Converse 1972]. The subjective well-being measurement scale involved questions regarding general satisfaction with life as a whole and its specific domains, including marriage, family, health, neighbours, friends and acquaintances, professional work, housework, leisure time, place of residence, living in the USA, living standards, income level and savings. The extent of satisfaction with each of the domains was evaluated in a seven-level scale, where 1 stood for very high dissatisfaction and 7 for very high satisfaction with a specific domain. The remaining points of the scale referred to moderate satisfaction or dis-

satisfaction levels described as a neutral state. The total QoL indicator was generated by adding up ratings for particular domains (the Crombach index for global satisfaction and its constituent life domains $\alpha$=0.81). The higher the index, the higher satisfaction level of a respondent.

The key to the success of the new QoL measurement tool lied in its simplicity and precedence ascribed to human life in the value hierarchy considered in the biological, social and spiritual dimensions. The change in the way of perceiving human life came to be reflected in economic, social and political decision taken at higher levels. Currently, QoL measurements are used to evaluate costs and profits of various health care programmes and medical interventions, distribution of social services, in particular those directed to the elderly and disabled persons and related to social needs and health care; distribution of financial resources between sectors of public life; as a legal basis for decisions concerning medical intervention taken to save life or lengthen survival and many other areas of social life.

## Concept of the quality of life – comments on methodology

### Difficulties in defining QoL

To begin with, it has to be noted that no universal or commonly acceptable definition of the quality of life (QoL) has been developed over nearly 60 years of scientific research in this area. What is more, the literature has introduced many new terms deemed as equivalent to QoL, such as: well-being, social well-being, social welfare, human development, happiness and life satisfaction, self-esteem, self-concept, self-efficacy, self-mastery, perceived autonomy and control, to mention just those with the largest number of records in the PubMed database. The diversity in the ways of understanding the concept of QoL has led to methodological chaos and difficulties in comparing study results. The concept has also proved to be highly ambiguous. This ambiguity may be found across various scientific disciplines. For example, in economics the quality of life is considered through the spectrum of economic well-being described by indexes and indicators showing to what extent material needs of households and communities are satisfied. Portfolio of goods and services, minimum income, level of consumption, extent of basic needs and social benefits are taken as reference points [Kamerschen et al. 1999]. Also in this respect, many various approaches to QoL, and consequently many different measures and indicators, can be identified. Another material measure of well-being is Human Development Index (HDI), introduced in 1990 by the United Nations to enable comparison of social and economic development levels of particular countries [http://hdr.undp.org]. The most popular non-financial measure of social well-being, reflecting both the management of natural resources and fair distribution of social goods for future generations is the Index of Sustainable Eco-

nomic Welfare (ISEW) [Daly and Cobb 1989]. This index is based on mean consumption, distribution of goods and degree of environmental degradation. The evaluation of economic well-being also employs a set of indicators reflecting some cause and effect relationships. The indicators have been developed by the UN Sustainable Development Commission under the Agenda 21 document [www.un.org/].

The quality of life in sociology is considered in the context of social well-being, which depends on the extent of meeting needs of existential nature related to safety and social contacts. Social well-being is determined by social standards, system of values and principles of social life. With framework of the analysed concept being so general, there are at least as many definitions of quality of life in sociology as there are research methodologies [Schuessler and Fisher 1985; Ferries 2004].

In psychology, the quality of life is understood as an emotional well-being associated with satisfaction and happiness of individuals and social groups [Bańka 2005]. The quality of life in this perspective relates to how every day reality is perceived and evaluated by an individual. It depends on the level of satisfaction with particular domains of life and life as a whole.

Health-related quality of life is understood as an ability of individuals or social groups to recognise, determine and fulfil their aspirations, as well as to satisfy their needs and select an environment to live in according to one's preferences, leading to the accomplishment of a fully satisfactory physical, mental and social well-being [Romney 2002]. In the case of illness, the quality of life is defined as patient's ability to evaluate functional effects of the disease and progress in treatment.

Looking for reasons behind the ambiguity of the concept of QoL, Farquhar indicates that some of them may stem from the difference in the way the concept is used by both specialists and laypersons [Farquhar 1995]. In common understanding, the quality of life has a broad meaning, mostly based on a positive association of the word 'quality'.

Specialist research focuses on explaining basic properties and the essence of QoL indicating whether it is positive or negative. But here again the concept of the QoL appears to be ambiguous [Constanza et al. 2008]. Rejeski and Mihalko [2001], reviewing publications on health, note that the concept of QoL used in those publications refers mostly to the psychological dimension. They add a critical comment arguing that many studies use the concept merely as a pretence to achieve specific study objectives.

Davis and colleagues [2006] reviewed paediatric literature with regard to QoL and HRQoL assessment instruments applicable to children aged 0 to 12 years. They also analysed the definitions of QoL and HRQoL. They found that QoL is understood as: position in life, functioning, functioning and feelings about functioning, existence, measured objectively or subjectively, the discrepancy between actual and ideal self. Whereas HRQoL as: functioning, functioning and feelings associated with functioning, functioning and well-being, health and feelings about health, a component of health, value assigned to duration of life [Davis et al. 2006: 315].

The above discussed disparities in understanding the concept of QoL are by no means exhaustive and yet sufficient to provide a good idea of the extent of the problem to be coped with while studying quality of life.

## Definition of the quality of life

Box 1 shows, in a chronological order, selected definitions of quality of life and well-being.

Box 1. Definitions of quality of life (QOL) and health-related quality of life (HRQOL)

| Definition | Author |
|---|---|
| Well-being stems from the degree of fit between individuals' perceptions of their objective situations and their needs, aspirations or values. | Campbell 1976 |
| The satisfaction of an individual's values, goals and needs through the actualisation of their abilities or lifestyle | Emerson 1985<br>quoted in Felce and Perry 1995:58 |
| A multi-faceted construct that encompasses the individual's behavioural and cognitive capacities, emotional well-being, and abilities requiring the performance of domestic, vocational, and social roles | Tartar et al. 1988<br>quoted in Meeberg 1993:33 |
| Quality of life is a feeling of overall life satisfaction, as determined by an individual whose life is being evaluated. Other people, preferably those from outside that person's living situation, must also agree that the individual's living conditions are not life-threatening and are adequate in meeting that individual's basic needs. | Meeberg 1993:37 |
| QOL is experienced when a person's basic needs are being met and when he or she has the opportunity to pursue and achieve goals in major life settings.<br>The QoL of an individual is intrinsically related to the QoL of other persons in her or his environment... QOL of a person reflects the cultural heritage of the person and of those surrounding him or her. | Goode 1994<br>quoted in Rapley 2003 |
| Quality of life is defined as an overall general well-being that comprises objective descriptors and subjective evaluations of physical, material, social and emotional well-being together with the extent of personal development and purposeful activity, all weighted by a personal set of values. | Felce and Perry 1995:60 |
| Quality of life is defined as an individual's perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards and concerns.<br>It is a broad-ranging concept incorporating in a complex way the person's physical health, psychological state, level of independence, social relationships, personal beliefs and their relationship to salient features of the environment. | WHOQOL Group 1995 |

| | |
|---|---|
| This definition reflects the view that QOL refers to a subjective evaluation which is embedded in a cultural, social, and environmental context. As such, QOL cannot be simply equated with the terms "health status", "life-style", "life satisfaction", "mental state", or "well-being". Rather, it is a multidimensional concept incorporating the individual's perception of these and other aspects of life. | WHOQOL Group 1995 |
| Quality of life and more specifically, 'health-related quality of life' refer to the physical, psychological, and social domains of health, seen as distinct areas that are influenced by a person's experiences, beliefs, expectations and perceptions (which we refer to here collectively as 'perceptions of health'). Each of these domains can be measured in two dimensions: objective assessments of functioning or health status, and more subjective perceptions of health. | Testa et al. 1996:835 |
| Quality of life is both objective and subjective, each axis being the aggregate of seven domains: material well-being, health, productivity, intimacy, safety, community and emotional well-being. | Cummins 1997 |
| QOL is a multidimensional evaluation of an individual's current life circumstances in the context of the culture in which they live and the values they hold. QOL is primarily a subjective sense of well-being encompassing physical, psychological, social and spiritual dimensions. In some circumstances, objective indicators may supplement or, in the case of individuals unable to subjectively perceive, serve as proxy assessment of QOL. | Haas 1999b |
| Quality of life is a concept that reflects a person's desired conditions of living related to eight core dimensions of one's life: emotional well-being, interpersonal relationships, material well-being, personal development, physical well-being, self-determination, social inclusion, and rights. | Schalock 2000:121 |
| Quality of life encompasses the basic conditions of life such as adequate food, shelter, and safety plus life enrichers such as inclusive social, leisure, and community activities. These enrichers are based on the individual's values, beliefs, needs and interests. | Schalock and Parmenter 2000 |
| A conscious cognitive judgement of satisfaction with one's life. | Rejeski and Mihalko 2001:23 |
| Quality of life is multidimensional in construct including physical, emotional, mental, social, and behavioural components. | Janse 2004:654 |
| Quality of Life (QOL) is the extent to which objective human needs are fulfilled in relation to personal or group perceptions of subjective well-being *(SWB, figure 1)*. Human needs are basic needs for subsistence, reproduction, security, affection, etc. *(see figure 1)*. SWB is assessed by individuals' or groups' responses to questions about happiness, life satisfaction, utility, or welfare. The relation between specific human needs and perceived satisfaction with each of them can be affected by mental capacity, cultural context, information, education, temperament, and the like, often in quite complex ways. Moreover, the relation between the fulfillment of human needs and overall subjective wellbeing is affected by the (time-varying) weights individuals, groups, and cultures give to fulfilling each of the human needs relative to the others. | Costanza R., Fisher B., Ali S., Beer C., Bond L., Boumans L., Danigelis N.L., Dickinson J., Elliott C., Farley J., Gayer D.E., MacDonald Glenn L., Hudspeth Th.R., Mahoney D.F., McCahill L., McIntosh B., Reed B., Rizvi A.T., Rizzo D.M., Simpatico Th., Snapp R. 2008 |

The review reveals once again the problem of multiple meanings of QoL. At the same time, however, the above indicated definitions show many common features, like the reference to specific domains of health (physical, psychological, social, emotional), multidimensional evaluation reflecting objective conditions and subjective well-being, favourable or unfavourable impacts of life conditions and positive or negative evaluation of those impacts.

Quality of life is set within a cultural context and, although some values can be regarded as universal, there is no universal standard that could be attributed to all cultures. The quality of life is thus a comparative rather than a direct designation. Gender, social status, age and generation play a very important role in determining QoL alongside with the cultural context, which seems to be the most significant factor in this respect [Schipper et al. 1988; Holmes 2005].

Farquar analysed definitions of QoL in relevant scientific publications [Farquhar 1995]. She classified all definitions into four categories: global, component, focused and combination. Farquar argues that global definitions are the most common, general type of definitions which speak little or nothing of components of QoL. They mostly deal with well-being and rely on the sense of satisfaction/dissatisfaction and happiness/unhappiness. The definitions proposed by Campbell and Emmerson (see Box 1) may be classified into the global category. The second type, component definitions, takes into account the components of the concept, dimensions, domains of life, or other characteristics that are deemed relevant to the quality of life. They involve evaluation of QoL both in a general dimension and in particular areas of human activity, as in the definition stated by Schalock (Box 1). Focused definitions refer to a single domain or several selected domains of life, e.g. health. This type of definitions is illustrated by the proposal of Testa et al. and the WHOQOL Group (Box 1). The last category distinguished by Farquar is that of combination definitions. They contain components of global and complex definitions, as well as aspects related to the environment, organisation of social life, emotional state, expectations, etc. The example of these is the definition proposed by Haas (Box 1).

In another systemic review of publications on various aspects of QoL, Taillefer and colleagues identified three types of QoL models: the conceptual model, conceptual framework and theoretical framework [Taillefer et al. 2003]. The conceptual model, the least sophisticated of the three, specifies dimensions and properties of QoL. The conceptual framework describes, explains or predicts the nature of the directional relationships between elements or dimensions of QoL. This approach to QoL can be found in the model proposed by the Centre of Health Promotion, University of Toronto, presented in Box 2 [www.utoronto.ca].

The proposal refers to the QoL construct proposed by Raeburn and Rootman [Raeburn, Rootman 1996]. Its graphical representation is shown in Figure 1. The model consists of specific components of health and well-being. It is based on a subjective state of enjoyment experienced by an individual, as expressed by the "degree to which a person enjoys the important possibilities of his or her life". Life possibilities arise from opportunities and limitations that an individual has in his or her life and reflect interactions between personal predispositions and environ-

Box 2. Quality of life model proposed by the Centre of Health Promotion, University of Toronto

| Being | Who we are? |
|---|---|
| physical | – physical health |
| | – personal hygiene |
| | – nutrition |
| | – experience |
| | – body care and clothes |
| | – general physical appearance |
| psychological | – mental health and adaptation |
| | – cognition |
| | – feelings |
| | – self-dignity, self-esteem |
| spiritual | – personal values |
| | – personal standards |
| | – beliefs |
| Belonging | Relationships with immediate environment |
| physical | – home |
| | – school and work |
| | – neighbourhood |
| | – community |
| social | – compatriots |
| | – family |
| | – friends |
| | – workmates |
| | – neighbours and local community |
| social | – adequate income |
| | – health and social services |
| | – employment |
| | – educational programmes |
| | – recreational programmes |
| | – community activities and events |
| Becoming | Pursuing personal goals, hopes and aspirations |
| activities | – housework |
| | – paid work |
| | – school or voluntary activities |
| | – health and social needs |
| leisure | – activities promoting relaxation and stress reduction |
| development | – activities supporting maintenance and improvement of knowledge and skills |
| | – adaptation to change |

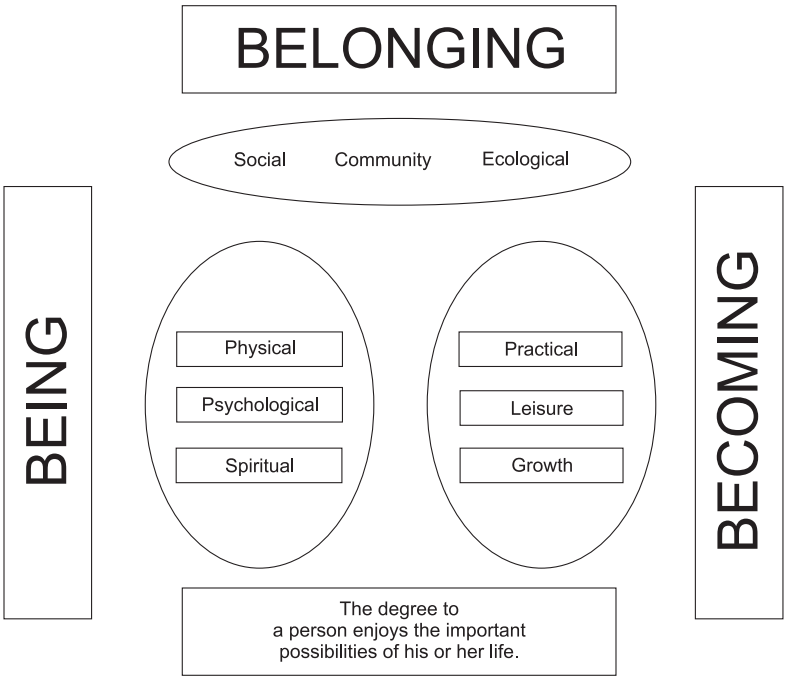



Fig. 1. Graphical presentation of the QoL construct proposed by Raeburn and Rootman [1996]. See text for explanation

mental factors. Enjoyment is comprised of two constituents: experiencing satisfaction and having certain desired properties, such as good health, as expressed in a phrase: "She enjoys a good health". The conceptual framework encompasses three dimensions: being, belonging and becoming. A set of factors enabling the experience of enjoyment was identified for each of the three.

The most sophisticated type of model is the theoretical framework. That approach includes a structure of the elements and their mutual relationship within a theory that explains these relationships, as the QoL model proposed by Langlois and Anderson [2002] and shown in Figure 2.

According to Meeberg, [1993], the quality of life is constituted by four key attributes: 1) sense of satisfaction with one's life, 2) capacity to assess one's life as being manifested with satisfaction or other feelings, 3) acceptable physical, mental, social and emotional health status, 4) objective evaluation of life conditions of a person made by others and making sure that they do not pose a life threat.

Concluding the review of QoL definitions, it needs to be stressed that the concept develops exponentially. One should agree with Haas (see Box 1) in claiming that the quality of life as a multidimensional evolution of existing life conditions of an individual in the context of culture and values that he or she respects and follows. The specificity of QoL applying to any of the proposed definitions lies in the fact that the evaluator is at the same time a subject evaluated, as individuals assess their lives according to their own criteria.

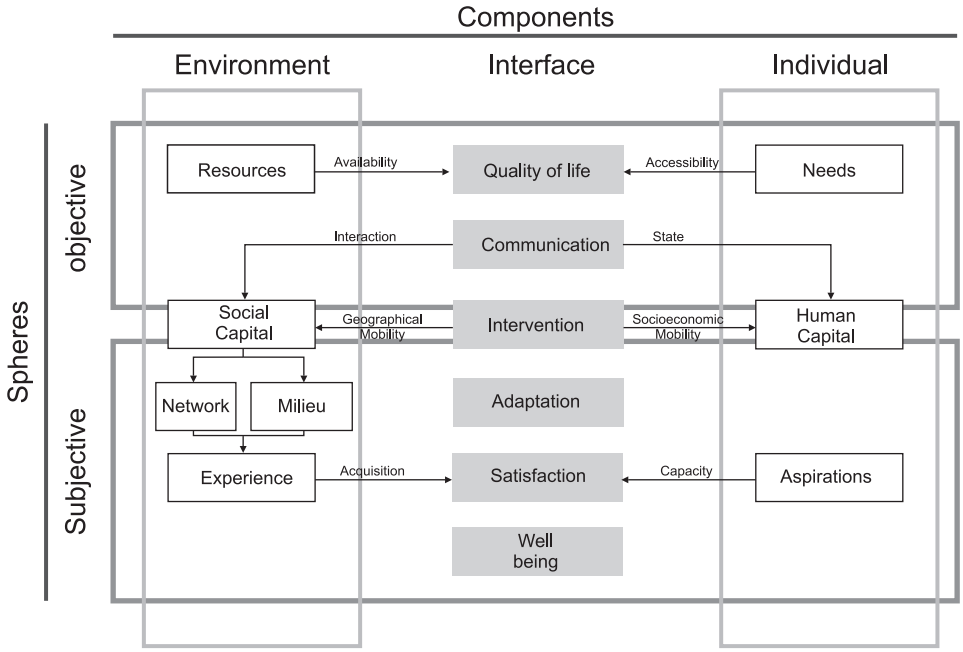



Fig. 2. Graphical presentation of the QoL model proposed by Langlois and Anderson [2002]. See text for explanation

Furthermore, the quality of life, as argued by Constanza and colleagues, is strictly related to well-being, human needs and the extent to which those needs are met. The human is not an isolated being, but part of a social group – a family, a peer community, association of interest, etc.

## Health-related quality of life

Health status, functional state and quality of life are the three concepts that are often used as equivalents of 'health' [Patrick and Bergner 1990; Seligman 2008]. The area of health stretches out between the negative aspects of life (with death at its most distressful end) and positive ones (performance of specific roles and sense of happiness). The choice of a concept depends usually upon the objective of a health evaluation and the gravity of the issue for patients, doctors and researchers. Doctors often rely on the assessment of biochemical and physiological parameters (objective measure of health) and seek to improve them as a way to improve a general health status. However, those parameters do not provide sufficient information of all aspects of health and, consequently, the efficacy of medical intervention. For example, they do not indicate patients' ability to function normally at home, in a family, at work or in society, nor their being free of pain and other physical and psychological disorders, or problems of social or financial

nature associated with the disease and its treatment. What is more, it turned out that conclusions based on observations of various aspects of health status are sometimes mutually contradictory. Therefore, the concept of QoL has been introduced into clinical examination. To indicate the association with health status a concept of health-related quality of life (HRQOL) has been adopted. It is wider than health status alone, as it takes into account factors that affect health status, such as income level, freedom, quality of the environment, factors related to health service and health care system. HRQoL allows to identify how a disease or its treatment relate to specific domains of life which are important to people in general (general HRQoL) and for those suffering from a specific disease (specific HRQoL). In clinical examination, QoL is mostly measured to evaluate changes induced by treatment. Elements subject to evaluation are self-reported health and general QoL [Guyatt et al. 1992; Cohen et al. 2006]. HRQoL is measured by behavioural and survival indicators which alter with physiological changes occurring in the organism. There are many arguments to support the application of HRQoL in health care. HRQoL is an important tool to manage patient flow and decision-making process with regard to an individual patient and health care in general. It is used to assess the impact of chronic diseases on patient condition, permits to evaluate the reasons why two patients suffering from the same disease respond differently to the same treatment, and allows to evaluate the effectiveness and economic implications of decisions regarding particular treatment modalities.

## Quality of life measurement

Stenner and colleagues indicated the most frequent issues to be tackled with in QoL measurement [Stenner et al. 2003]. These are: (1) a compound and multi-aspect definition of QoL hindering a quantitative/measurable determination of this complex construct; (2) subjective dimension of QoL dependent on the cultural context, gender, age, social class (it may happen that a questionnaire used will reflect the author's views and values rather than generally accepted systems of values); (3) there is no certainty that domains of life that a researcher will find important for QoL will also be important for an individual under study.

Let us start discussing these issues from determining domains of QoL. Schalock made a review of 9,749 abstracts, 2,455 articles and an in-depth review of 897 articles to propose eight principal domains of QoL along with their respective indicators and descriptions [Schalock 2004]. The eight domains of quality of life are: emotional well-being, interpersonal relationships, material well-being, personal development, physical well-being, self-determination, social inclusion, and rights. These domains are described the following indicators and indices:

- the emotional well-being is described by: contentment (satisfaction, moods, enjoyment), self-image (identity, self-worth, self-esteem), lack of stress (predictability, control):

- interpersonal relations are described by: interactions (social networks, social contacts), relationships (family, friends, peers), supports (emotional, physical, financial, feedback);
- material well-being is described by: financial status (income, benefits), employment (work status, work environment);
- personal development is described by: education (achievements, status), personal competence (cognitive, social, practical), performance (success, achievement, productivity);
- physical well-being is described by: health (functioning, symptoms, fitness, nutrition), activities of daily living (self care skills, mobility), leisure (recreation, hobbies);
- self-determination is described by: autonomy/personal control (independence), goals and personal values (desires, expectations), choices (opportunities, options, preferences);
- social inclusion is described by: community integration and participation, community roles (contributor, volunteer), social supports (support network, services);
- rights are described by: human rights (respect, dignity, equality) and fundamental legal (citizenship, access, due process).

Schalock assumed that out of 125 indicators used in 16 studies carried out in the 1990s, as many as 74.4% refer to the eight core QoL domains. Other proposed classifications of QoL domains are shown in Box 3. Three classifications (Cummins, Felce and Schalock) present QoL domains in studies regarding persons with disabilities. The other two concern health status (WHOQoL) and sociological research (Hagerty et al.). The number of separated QoL domains may be six (WHOQL Group and Felce), seven (Cummins and Hagerty et al.) or eight (as proposed by Schalock). All of the classifications reflect the material, physical, psychological and emotional aspects, as well as the aspect of being, belonging and becoming.

There are two types of instruments to measure health-related quality of life: generic instruments (which refer to overall quality of life) and condition-specific instruments (which refer to specific issues, for example a specific disease, group of patients or life function).

Quality of life measurement comprises two components: an objective and subjective one. The objective component refers to material conditions, socio-economic status, education, housing, neighbourhood, physical fitness and well-being. It is used to describe the nature of social changes and developmental processes [Bloom et al. 2001]. Other frequently used objective QoL indicators associated with social life include: life expectancy, quality of physical environment, crime rate, unemployment rate, poverty rate, school attendance, weekly working hours, infant mortality rate, mortality rate and suicide rate, and gross domestic product (GDP). The subjective component refers to the state of functional performance, well-being and satisfaction. In the social dimension, it is also about interpersonal relations, community activities, personal development and fulfilment, relaxation, social participation, safety, satisfaction with work, sexual life, perception of justice and identification of social class [Rapley 2003].

Box 3. Proposed classifications of QoL domains by various authors

| Source: | | | | |
|---|---|---|---|---|
| WHOQOL Group [1995] | Felce [1996] | Cummins [1997] | Hagerty et al. [2001] | Schalock [2004] |
| Study | | | | |
| Health | Disability<br>Psychology | Disability | Social Indicators | Disability<br>Psychology |
| Quality of life domains | | | | |
| Environment | material well-being | material well-being | material well-being | material well-being |
| Social relationships | social well-being | community well-being | belonging to local community | social inclusion |
| | productive well-being | productive work/activity | productive work and activity | |
| Physical | physical well-being | health | health | physical well-being |
| Psychological | emotional well-being | emotional well-being | emotional well-being | emotional well-being |
| | civil rights | | | rights |
| | | social/family relations | relationships with family and friends | interpersonal relationships |
| | | | | personal development |
| Level of independence | | | | self-determination |
| Spiritual | | safety | personal safety | |

The correlations between both components are not clear. This means that none of the objective QoL indicators explains any of the subjective components, and the other way round: none of the objective components can be derived from the subjective QoL indicators. It has been observed that the relevance of both components in evaluation of QoL is uneven. Objective indicators, such as socio-economic status, education, housing and neighbourhood, are estimated to account only for 15% of the total QoL variance, whereas subjective (psychological) indicators, such as happiness and satisfaction, for as much as 50% [Day, Jankey 1996]. While showing the importance of the subjective dimension of QoL, where the psychological context plays a key role, those figures do not undermine entirely the objective dimension.

According to Raphael [1996], objective QoL indicators serve to evaluate the functioning of a society and are instrumental in multidisciplinary studies, introduction of social standards, and development and implementation of social policy programmes. Subjective indicators, on the other hand, facilitate identification of the conceptual nature of QoL and are used for self-evaluation [Guyatt et al. 1992].

Constanza and colleagues [2008] proposed a holistic vision of QoL indicators. In their proposal, they combined the objective indicators with the subjective dimension. This approach appears to be the best way to measure the quality of life.

## Examples of health-related quality of life scales

The measurement of HRQoL is conducted by means of instruments based on measurement scales. They have the form of a questionnaire which is completed by patients themselves or by a surveyor interviewing patients. The questionnaire may be used in studies of cross-sectional or longitudinal design. Cross-sectional study results show the extent of variation of treatment outcomes in patients treated with the same method (variation/distinction instrument) They allow a division of patients by their current performance or prognosis. Results of longitudinal studies indicate changes in the effect of treatment observed in an individual patient or group of patients observed during an intervention (evaluation instrument). The effect of treatment is measured by the number of points scored in the questionnaire and in comparison with a control group of patients who have not been subjected to treatment. Evaluated aspects include patients' physical, psychological and emotional behaviours during treatment or rehabilitation programme and efficacy of treatment involving a new drug.

The adequacy of study results depends on the quality of instruments used to conduct those studies. Estimate (psychometrical) scales which form the basis for the QoL measurement should meet the following criteria: (1) validity, including face validity and construct validity, (2) reliability, and (3) responsiveness. The validity criterion means the ability of an instrument to measure processes occurring in domains in which researchers are really interested and which are specified in study objectives. Reliability is the ability of an instrument to carry out repetitive measurement in the same patient and to detect differences in patients who vary in terms of parameters measured. Responsiveness is an ability of an instrument to detect even the slightest differences in a patient.

The scales meeting the above quality criteria should enable doctors and decision-makers in the areas of health care and medical services to identify all differences, both those observed in a cross-sectional and long-term perspective, from those slight and hardly noticable to those clearly visible.

The selection of a QoL measurement instrument depends on the aim of a study, clinical context and aspects investigated. QoL measurement may serve to determine a general health status or preferences where there is a choice. It may involve screening tests aimed to spot a specific category of patients or to identify changes of an analysed parameter in time. It may be a study focused on a single aspect of QoL or concerning many aspects, if the evaluation requires a multi-level measurement. Measurement may apply to overall quality of life (generic instruments) or particular aspects which are specific for a given case (specific instruments). Overall QoL measurements employ so-called health profiles or instruments measuring the

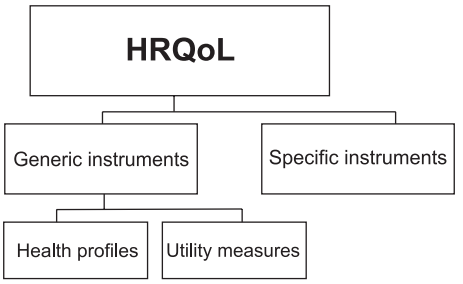



Fig. 3. The classification of HRQoL measures based on the proposal by Cramer and Spilker [1998]

utility of a health status, its value, satisfaction related to it, and contentment (utility measures). The classification of HRQoL measures based on the proposal by Cramer and Spilker [1998] is shown in Figure 3.

Spilker et al. [1990] established that there were 500 various proposals of HRQoL measurements available in 1990. Davies et al. [2006] mention at least 14 generic type instruments and 21 condition-specific QoL and HRQoL instruments used in paediatrics for treatment of children aged 0 – 12. An extensive discussion of currently used QoL and HRQoL evaluation instruments for adolescents and children can be found in reviews provided by Davies et. al. [2006] and Kaczmarek [2009].

## Final remarks

Based on the review of existing publications and discussions, we might venture to formulate a generalised concept of quality of life. In our view, it is a multifaceted category comprising physical, mental and social well-being of an individual and society. It is developed in conjunction with bio-geographical, socio-economical and health-related factors. Those factors in aggregate influence the development of an individual and society, by ways which are not yet fully understood. Quality of life or a well-being are possible to achieve by experiencing independence, having freedom of choice and free access to resources available in the environment. The above understanding of QoL reflects its multifaceted nature while referring to the intrapersonal (experiencing independence), interpersonal (having freedom of choice) and social (free access to resources available in the environment) levels. Quality of life thus comprehended as a normative concept, associated with systems of values, aspirations and expectations held by individuals or social groups they belong to, and with how an individual perceives his or her position in life and a specific cultural context. Psychologically, QoL means a person's sense of satisfaction and happiness. It is based on a personal experience and reflection on what we feel and what kind of people we are. The sources of our emotions are located in ourselves and in our environment, and subjective evaluation of our life is the result of experienced circumstances, representing a psychological aspect of QoL.

The hierarchy of life domains that are subject to self-evaluation changes with stage of life and experience. Indeed, the experience of a young person at the doorstep to adulthood is different than that of an adult or that of an elderly person.

An objective dimension of QoL serves to evaluate social inequalities and to predict the directions of social development by using physical, social and economic in-

dicators that reflect the material aspects of life. The objective QoL indicators include also health regarded as proper functioning of all biological systems. Subjective QoL indicators reflect a psychological well-being, sense of satisfaction or happiness arising from relations with other people rather than material goods. They determine the level of satisfaction or happiness resulting from a good health status or relations with other people. Subjective view of one's life is the outcome of internal processes of evaluating life as a whole or its particular domains. The order of importance of particular life domains is linked with life stage and experience. It depends on an individual system of values, in particular on the individual approach to the sense of life. This view is very close to the QoL concept proposed by Constanza et al. [2008: 18].

The period of adolescence is unique for many reasons, which have been mentioned many times in this book, also because it is in this period of developmental transformation, the transition from childhood into adulthood, that an individual system of values is developed to affect the quality of life of a young person.

## Quality of life of adolescents and underlying social and lifestyle behaviour factors: research findings

### Background

It is well recognized that many factors determine the health and development of children and adolescents. From among them, the standard of living and lifestyle behaviours have a crucial role to play in health outcomes and well-being. Adolescence is marked by increasing involvement in health risk behaviours, such as unhealthy eating, inadequate physical activity, substance use, and unsafe sexual behaviour continuing into later adult life. These risk-for health lifestyle behaviours have long-term effects in later life, and many of them have the most important associations with all cause mortality and morbidity in adulthood [Mini et el. 2006].

Association of lifestyle behaviours with other social determinants of health, involving standard of living, peer pressure and fear are believed to play a synergistic role in human health and quality of life. It is, therefore, important to identify these factors in adolescents and intervene early to improve living conditions and to alter patterns of behaviour that would place people at health risk in later life and in this way to improve a person's quality of life.

***Study objectives*** were to investigate socioeconomic and lifestyle choices as single predictors of self-perceived quality of life, considered as a broad measure of psychosocial well-being, during the period of adolescence (between the ages of 13 and 18 years). The specific objectives were: (1) to draw a pattern of relationships between SES and health-related behaviour, (2) to identify demographic, social and lifestyle behaviour predictors of adolescents' general quality of life, and (3) to estimate gender differences in QoL.

***The study hypothesis*** is that adolescents of low socioeconomic status (based on the SES and FAS II indicators) and unhealthy behaviours (based on the lifestyle health behaviour) are supposed to report significantly lower perceived QoL scores than their better-off and lifestyle healthy behaviour counterparts.

## Study design

This was self-administered cross-sectional survey available by paper-and-pencil and conducted during school time under the ADOPOLNOR project, between May 2009 and June 2010. The research was conducted with approval of the Bioethics Commission of Poznań University of Medical Sciences and Poznań Board of Education. Both parents or guardians and adolescents completed written consent or assent forms. Each participant of the survey was labeled with his/her own individual research (ID) number. Detailed description of the project design and surveys that have been carried out under the ADOPOLNOR project one may find in the chapter on "Conceptual framework..." in this volume.

Each student was asked to complete anonymously the YQOL-R survey and the ADOPOLNOR-M survey at one sitting lasting about 45 minutes each. The ADOPOLNOR-R survey was administered to the students' parents or responsible guardians. The survey took about 30 minutes of parents' time. Parents completed the survey within 2–3 days of receiving the paper copy.

Somatometric measurements were taken in a parallel auxological research survey and provided data on adolescents' weight and height.

## Methods

Data were collected from 2,758 students aged between 13 and 18 and their parents or responsible guardians. Of them, 1386 were girls and 1372 were boys.

Polish version (Opisując twoje życie) of the Youth Quality of Life Instrument – Research Version (YQOL-R) was applied to measure general quality of life (QoL) that encompasses of all aspects of psychosocial well-being. The Youth Disability Screener (YDS) items were used to determine young people's perceived health status and self-reported conditions that were causing long-term limitations in their functioning or activities.

The World Health Organization has described QoL as "individuals' perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards and concerns" [WHOQOL Group 1995: 1403]. This broad, generic conceptualization of QoL provides theoretical underpinning of the YQOL-R instrument. Topolski and colleagues state "QoL defined this way is broader and more global than the concept of 'subjective well-being' in reflecting the cultural and social context that defines *the good life*" [Topolski et al. 2002: 1].

The YQOL-R is a generic quality of life (QoL) measurement designed for all youth, ages 11–18, including those with and without disabilities [Edwards et al. 2002; Topolski et al. 2002; Edwards et al. 2003]. It is an easy-to-understand self-administered questionnaire designed to assess perceptual (i.e., known only to the youth themselves), contextual (i.e., can be reported by others, and as such potentially verifiable), and individual specific (i.e. top 5 facets most important to individual and 5 facets they would like to change for the better) aspects of quality of life with respect to 'self', 'relationships', 'environment' and 'general quality of life'. The length of contextual aspect is 15 items; the length of perceptual aspect is 41 items. In addition to a total score, four domains have been identified from the YQOL-R. There are:

1) The Sense of Self domain pertains to adolescents feelings about themselves. Several subcategories comprise this domain including: belief in self, being oneself, mental health, physical health, and spirituality.
2) The Social Relationships domain pertains to the adolescent's relations with others, and has following subcategories: adult support, caring for others, family relations, freedom, friendships, participation, and peer relations.
3) The Environment (Culture and Community) domain pertains to opportunities and obstacles in the adolescent's broader social and cultural milieu. Subcategories include: engagement and activities, good education, liking neighbourhood, monetary resources, personal safety, and view of the future.
4) The General Quality of Life domain pertains to the adolescent's sense of how well his or her life is going overall. Subcategories include: enjoying life, feeling life is worthwhile, and being satisfied with one's life.

The YQOL-R scores are transformed to a 0–100 scale for easy interpretability, higher scores indicating better QoL.

The ADOPOLNOR-M is a 61-item semi-structured survey instrument, designed for adolescents as an extended version of the HBSC protocol, and was used to assess health-related behaviours such as smoking cigarettes, substance abuse, habitual physical activity and sexual behaviour.

The ADOPOLNOR-R is a 26-item closed-format (multi choice questions) survey questionnaire designed for parents. It was used to obtain information on parents' personal characteristics (demographic, education, profession and occupation status), social and economic indicators of standard of living including financial strain, lifestyle behaviour, retrospective and current information on their own child's disabilities and/or diseases.

Socioeconomic status (SES) was measured in two ways, using: (1) traditional SES indicators (such as resident area, mother's education level, and family finance-related burden referred to as *financial strain* indicative of the objective financial situation); this information was provided by adolescents' parents (via the ADOPOLNOR-R survey instrument), and (2) perceived family wealth reported by adolescents themselves, the adolescents' SES, using the Family Affluence Scale II (FAS II).

A question battery, the Family Affluence Scale II (FAS II), originally based on the work of Peter Townsend [1987], was further developed by the WHO Health Be-

haviour in School-Aged Children Study (HBSC) as an alternative measure to parental occupation information and perceived family wealth collected from parents themselves [Curie et al. 1997].

The FAS II, a four-item measure of family wealth, provided by students was measured by:

- number of cars in family: no (0); yes, one (1); yes, two or more (2);
- asking if the respondent have one's own bedroom: no (0); yes (1);
- number of family's vacation travels during the past 12 months: not at all (0); once (1); twice (2); more then twice (3);
- number of computers in household: none (0); one (1); two (2); more than two (3).

The FAS II total score could range from 0 to 9, with higher scores indicating higher level of the family wealth. It may be scored as a composite score and classified into three categories of low, middle and high level of family wealth.

A Modifiable Activity Questionnaire for Adolescents (MAQA) instrument, adapted from the Youth Risk Behaviour Survey Questionnaire (YRBSQ), was used to assess adolescents' habitual physical activity including a selection of sedentary activities such as television, video, computer and others [Aaron et al. 1995]. Physical activity before, during and after school as well as on weekends was reported as a 7-day recall of physical activity lasting at least 30 minutes per day. First, the total time (in hours) per week spent on various types of activity was assessed. The Metabolic Equivalent Task (MET)/per hour score was subsequently derived from the time assessment as a measure of the intensity of a physical activity. MET is a unit used to compare the working metabolic rate (the amount of oxygen used by the body during physical activity) to the resting metabolic rate of 1.0 (4.184 kJ)/kg/h. One MET is defined as 1 kcal/kg/hour and is roughly equivalent to the energy cost of sitting quietly. MET is also defined as oxygen uptake in ml/kg/min with one MET equal to the oxygen cost of sitting quietly, equivalent to 3.5 ml/kg/min. Activities are listed in the Compendium of Physical Activities as multiples of the resting MET level and range from 0.9 (sleeping) to 18 METs (running at 10.9 mph) [Ainsworth et al. 1993; 2000; 2011]. For example, at rest, the body uses 1 MET for basic functions such as breathing, brisk pacing at home 2 METs; brisk walking in a hurry 3 to 4 METs; catching a bus while carrying a small luggage 5 to 6 METs; jogging and sprinting higher than 6 METs and so on. Describing an activity using MET allows comparisons among people of different weight. To get weekly MET scores, MET value for each activity was multiplied by hours expended in that activity each time, then added all weekly activities.

Chronological age was calculated in decimal values by subtracting date of examination from the date of birth. The age groups were divided by years, defined in terms of the whole year. The initiation of sexual activity was assessed based on information obtained from participants who reported whether they ever had sexual intercourse with female (reported by boys) or male (reported by girls).

Stature was measured with a GPM anthropometer to the nearest millimeters according to the technique described by Knussman [1988]. Body weight was measured with a scale precise to ± 100 g.

Body Mass Index (BMI) was calculated using the BMI formula (dividing body weight in kilograms by body height in square meters) to find the height to weight ratio of an individual. Using Cole's cut off points, individuals' weight status was classified into 4 categories: underweight, normal weight, overweight and obesity [Cole 2000, 2007].

***Data analysis*** was based on 2,758 students' questionnaires with complete information on the variable of interest that is accounted for final response rate of 85.1%. The final response rate of parents was 81.7%.

The outcome of interest was the assessment of quality of life of adolescent girls and boys and the variables hypothesized as covariates of QoL were indicators of demographic, socioeconomic status and cultural/lifestyle behaviour factors. The dependent variable was a quantitative Quality of Life Total Perceptual Score (QoLTPS). The QoLTPS was analyzed in relation to demographic, socioeconomic, lifestyle behaviour and health variables.

The variables in question were quantitative (continuous) and qualitative, categorized as binary or multi-state categorical variables, with each of $k$ states representing a separate group of independent variables. Quantitative independent variables included chronological age and BMI. For purposes of this study, chronological age groups were divided by years and classified into two age groups corresponding to school type, 13–15 years old students of junior secondary school and 16–18 years old students of senior secondary school.

Body Mass Index (BMI) was classified in 4 categories: underweight, normal weight, overweight and obese.

Qualitative independent variables were dummy variables for socioeconomic status, lifestyle behaviour and health-related factors. Residual area was binary categorized to rural; urban. Mother's educational attainment, reported in years of completed schooling or as a level of formal education, was classified in three schooling categories: (1) low level – primary/vocational (7–11 years attained); (2) medium level – secondary school lasting 12 years; (3) high/academic level lasting more than >12 years.

Financial strain (reported by parents/guardians) was measured based on family's difficulty with respect to affording food, clothing, housing, car, furniture, leisure activities, and money owed. Items were rated on a binary scale: (1) no difficulty; (2) difficulty.

The FAS II total score could range from 0 to 9, with higher scores indicating higher level of the family wealth. In the study, it was scored as a composite score and classified into three categories: low affluence (0–2), middle affluence (3–5) and high affluence (6–9). Those cases who failed to respond to any one or more FAS II items were categorized as Indeterminate.

Smoking status was reported as: (1) never smoked; (2) former smoker, (3) current smoker.

The intensity of habitual physical activities, expressed in terms of MET, was classified in three levels: (1) low - equivalent to <3.0 METs/week; (2) moderate 3.0–6.0 METs/week; and (3) vigorous >6.0 METs/week.

Sexual debut was defined as (1) yes and (2) not yet.

Self-rated health was assessed using a 5-grade Likert scale ranging from poor to excellent.

The Youth Disability Screener (YDS) had 5 items on self-reported disability answered either *yes* or *no*. Adolescents who endorsed any one of the 5 items as a *yes* were considered to have a self-reported disability.

The data from the questionnaire were coded and entered into a data file, and combined with data on somatometrics. All statistical computations were performed using STATISTICA data analysis software system, version 9.0 (2009) www.statsoft.com.

Descriptive statistics were first computed to provide a general picture of the sample. Chi-square tests (Fisher's exact test) were used to make comparisons between girls and boys in relation to categorical variables (including younger and older age groups of adolescents corresponding to the type of school), resident areas, mother's education level, financial strain of the family, family affluence FAS II, smoking status, habitual physical activity, sexual debut, weight status, perceived health and disability), and *F*-test was used to determine significant differences between variables at 95% confident interval.

A structure of categorical independent variables on the socioeconomic and lifestyle behaviour factors was analysed using a Multiple Correspondence Analysis (MCA) model. The MCA is a multivariate exploratory/descriptive data analytic technique designed to analyze multi-way tables containing some measure of correspondence between the rows and columns. MCA may be considered to be an extension of simple correspondence analysis (CA) to more than two variables and for easier interpretation often discuss the simple CA of an indicator or design matrix. As opposed to traditional hypothesis testing designed to verify *a priori* hypotheses about relations between variables, exploratory data analysis is used to identify systematic relations between variables when there are no (or rather incomplete) *a priori* expectations as to the nature of those relations. An important feature of correspondence analysis is the multivariate treatment of the data through simultaneous consideration of multiple categorical variables [Quoted: 6.5. Correspondence Analysis. Available at http://www.unesco.org/webworld/idams/advguide/Chapt6_5.htm; Accessed March 20, 2011].

The graphical representation of column profiles was used for detecting structural relationships among categorical variables of particular SES and lifestyle factors. Each of these factors was then evaluated as potential predictor for the quality of life. First, using a one-way analysis of variance ANOVA, influence of one treatment factor with two or more treatment levels on the quality of life expressed in terms of the QoLTPS means was assessed. The Tukey's HSD (Honestly Significant Difference) test, used in conjunction with ANOVA, enabled us to find which means were significantly different from one another. Then, the study hypothesis was tested using uni- and multivariate analyses of covariance ANCOVA, with socioeconomic and lifestyle behaviour factors as the main effect predictive variables to QoL, and with age as covariate.

## Results

*Sample characteristics* are summarized in Table 1.

Of the eligible 2,758 students, 49.7% were females and 50.3% males. There were no significant differences between boys and girls in terms of age ($\chi^2$=1.3 df=1 N=2758 *NS*), rural and urban resident places ($\chi^2$=1.6 df=1 N=2758 *NS*) and mother's education level ($\chi^2$=2.0 df=2 N=2758 *NS*). A significantly larger proportion of girls' parents reported living under economic pressure (14.1% as compared with boys' parents 10.5%); however, an overwhelming majority (89.5% of boys' and 85.9% of girls' parents) reported having no financial problems. Both adolescent boys and girls were likely to live in families of similar affluence. Similar proportions of boys and girls reported low (14.3% compared with 14.2%) as well as

Table 1. Sample characteristics

| Variable | | Males N=1372 | | Females N=1386 | |
|---|---|---|---|---|---|
| | | n | % | n | % |
| Age group (years) | 13–15 | 617 | 44.9 | 644 | 46.5 |
| $\chi^2$=1.3 df=1 NS | 16–18 | 755 | 55.1 | 742 | 53.5 |
| Resident area | Rural | 574 | 41.8 | 546 | 39.4 |
| $\chi^2$=1.6 df=1 NS | Urban | 798 | 58.2 | 840 | 60.6 |
| Mother education | 7–11 years | 535 | 39.0 | 579 | 41.8 |
| level (years attained) | 12 years | 558 | 40.7 | 538 | 38.8 |
| $\chi^2$=2.0 df=2 NS | >12 years | 279 | 20.3 | 269 | 19.4 |
| Financial strain | Yes | 144 | 10.5 | 195 | 14.1 |
| $\chi^2$=7.8 df=1 $p$<0.01 | No | 1228 | 89.5 | 1191 | 85.9 |
| FAS II | Low | 196 | 14.3 | 197 | 14.2 |
| (affluence level) | Middle | 759 | 55.3 | 762 | 55.0 |
| $\chi^2$=0.03 df=2 NS | High | 417 | 30.4 | 427 | 30.8 |
| Smoking status | Current | 189 | 13.8 | 184 | 13.3 |
| $\chi^2$=2.4 df=2 NS | Former | 96 | 7.0 | 89 | 6.4 |
| | Never | 1087 | 79.2 | 1113 | 80.3 |
| Physical activity | Low | 49 | 3.6 | 128 | 9.2 |
| (METs/week) | Moderate | 556 | 40.5 | 739 | 53.3 |
| $\chi^2$=97.7 df=2 $p$<0.01 | Vigorous | 767 | 55.9 | 519 | 37.5 |
| Sexual debut | Yes | 255 | 18.6 | 181 | 13.1 |
| $\chi^2$=12.3 df=1 $p$<0.01 | Not yet | 1117 | 81.4 | 1205 | 86.9 |
| BMI (kg/m$^2$) | Overweight and Obese | 243 | 17.7 | 173 | 12.4 |
| $\chi^2$=22.9 df=2 $p$<0.01 | Underweight | 112 | 8.2 | 167 | 12.1 |
| | Normal | 1017 | 74.1 | 1046 | 75.5 |
| Self-rated health | Poor | 22 | 1.6 | 48 | 3.5 |
| $\chi^2$=101.6 df=4 $p$<0.01 | Fair | 60 | 4.4 | 112 | 8.1 |
| | Good | 332 | 24.2 | 473 | 34.1 |
| | Very good | 630 | 45.9 | 574 | 41.4 |
| | Excellent | 328 | 23.9 | 179 | 12.9 |
| Youth disability | Yes | 342 | 24.9 | 406 | 29.3 |

high levels (30.4% compared with 30.8%) of family affluence ($\chi^2$=0.03 df=2 N=2758 *NS*).

With regard to lifestyle behaviour, it was found that of the respondent boys and girls, about 20% were either currently smoking (14.3%) or had quit smoking (7%). The vast majority however reported that they had never smoked (about 80%).

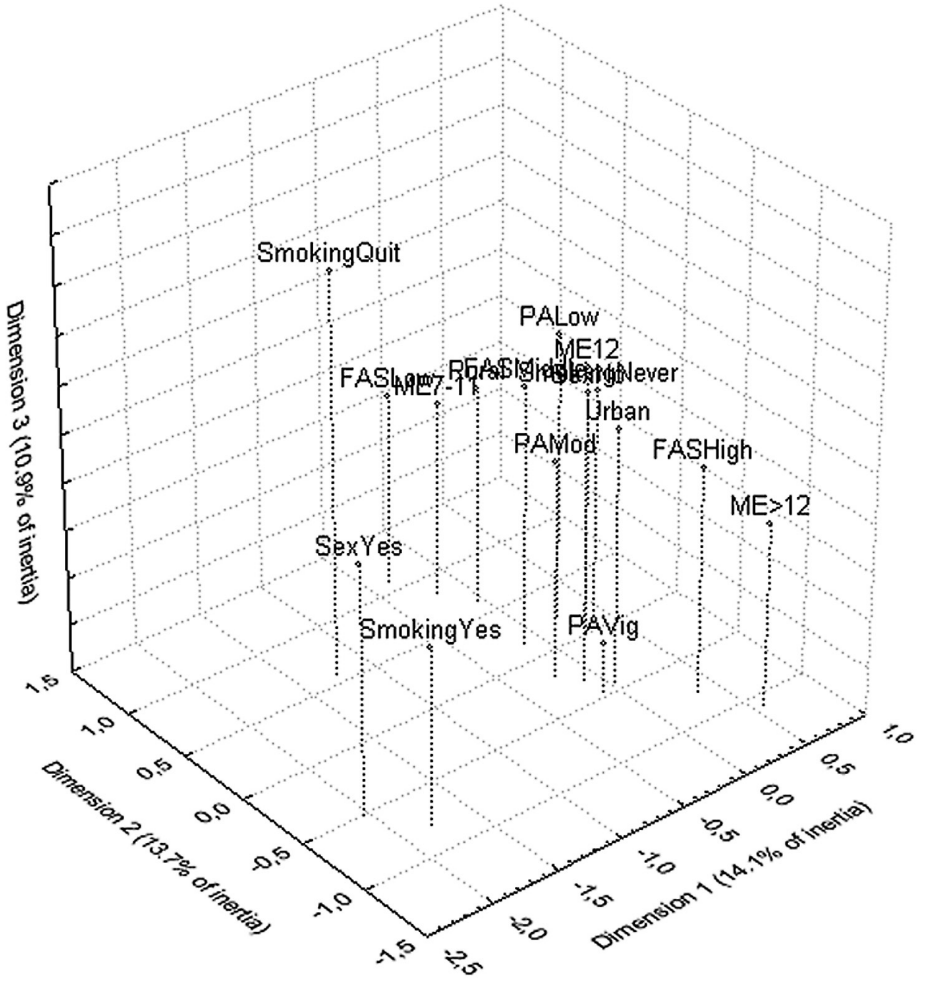



Fig. 4. Three-dimensional plot of column co-ordinates; dimension 1×2×3. Input Table (rows*columns): 16×16 (Burt's Table) of the SES and lifestyle behaviour factors. The following factors were analyzed and abbreviation used: (1) Residence area (Rural; Urban); (2) Mother educational attainment: primary and/or vocational (ME7-11); secondary (ME12), and high/academic (ME>12); (3) FASII: low (FASLow), middle (FASMiddle), and high (FASHigh); (4) Smoking status: current smokers (SmokingYes), former smokers (SmokingQuit), and never smoked (SmokingNever); (5) Intensity of habitual physical activity: low (PALow), moderate (PAMod), and vigorous (PAVig); (6) Sexual debut: yes (SexYes), no (SexNo). See text for further explanation of the MCA model

Boys were more likely than girls to be engaged in vigorous-intensity physical activity (55.9% compared with 37.5%). It is therefore surprising to find that a greater proportion of boys than girls (17.7% compared with 12.4%) was classified as overweight and/or obese.

There were also significant gender differences in self-perceived health. The proportion of boys reporting excellent health was twice that of girls (23.9% as compared with 12.9%). In opposite, the proportion of girls reporting poor health was twice that of boys (3.5% as compared to 1.6%). Overall, majority of boys and girls were likely to evaluate their health positively either good or very good.

*Structural relationships* among the socioeconomic and lifestyle health behaviour factors are shown in three-dimensional asymmetric plot in Figure 4.

The design matrix of MCA contained the SES and lifestyle factors categorical variables that would be interpreted in specified category space. The input table (Burt's table) involved rows (cases) and columns (categories). The cases included: resident area (2 categories), mother's education level (3 categories), FASII (3 categories), smoking status (3 categories), sexual debut (2 categories) and intensity of habitual physical activity (3 categories). A 16×16 row and column profile matrix was achieved to scaling the coordinates. These coordinates were plotted in a three-dimensional scatterplot along with the column coordinates. The final display represented the relationships (similarities) among such factors as SES, lifestyle behaviour and perceived family wealth. The goodness of fit of the model, estimated by chi-square value, was: the total value of $\chi^2 = 25757.4$ df = 225, and $p = 0.00$.

The total inertia, defined as the total Pearson Chi-square for the multi-way divided by the total sum 2,758 in the present sample, yielded 1.7. The first and second axes accounted for 14.1% and 13.7%, and the third one for 10.9% of the inertia, respectively. A cumulative total, often referred as to the retention solution, accounted for 38.7% of the total inertia.

With regard to first dimension, the vast majority of variables were scattered near the origin of axes. The most rightwards, with positive value coordinates (+0.58) were adolescents who highly assessed family affluence and whose mothers attained high/academic level of education. In the opposite site, most leftwards with negative value coordinates (–2.04), were current smokers who had already made the sexual debut. The group of former smokers markedly outlay from others.

The second dimension enabled us to identify the clustering structure of socioeconomic and lifestyle factor variables. Rural residence was closely related to low level of maternal education and low adolescents' perceived family affluence (first cluster). There were associations among adolescents' middle family affluence, low physical activity, never smoked status and not making sexual debut; urban residence was associated with moderate level of maternal education and moderate to vigorous physical activity (second cluster). High level of maternal education was related to high perceived family affluence (third cluster). Smoking habit was associated with making sexual debut (fourth cluster).

The quality of solution was judged using auxiliary statistics such as coordinates of the first, second and third dimensions, mass, quality, relative inertia, relative iner-

Table 2. Quality of Life Total Perceptual Score sorted by gender, socioeconomic and lifestyle behaviour factors

| Variable | Males | | | | Females | | | |
|---|---|---|---|---|---|---|---|---|
| | n | Mean (95%CI) | SD | *F* | n | Mean (95%CI) | SD | *F* |
| Age group | | | | | | | | |
| 13–15 years | 617 | 78.2 (77.1–79.4) | 13.4 | 0.7 | 644 | 78.2 (76.9–79.4) | 14.5 | 8.3 |
| 16–18 years | 755 | 78.9 (77.9–79.9) | 13.2 | NS | 742 | 75.7 (74.7–76.8) | 14.4 | ** |
| Resident area | | | | | | | | |
| Rural | 574 | 78.6 (77.3–79.8) | 13.9 | 0.3 | 546 | 77.3 (75.9–78.6) | 14.3 | 0.8 |
| Urban | 798 | 78.5 (77.6–79.4) | 12.9 | NS | 840 | 76.5 (75.4–77.6) | 14.6 | NS |
| Mother education | | | | | | | | |
| 7–11 years | 535 | 77.7 (76.4–78.9) | 13.5 | 2.1 | 579 | 76.5 (75.1–77.9) | 15.0 | 0.3 |
| 12 years | 558 | 78.4 (77.2–79.7) | 13.4 | NS | 538 | 76.8 (75.5–78.1) | 14.3 | NS |
| >12 years | 279 | 79.9 (78.3–81.5) | 12.1 | | 269 | 77.3 (75.6–79.1) | 13.6 | |
| Financial strain | | | | | | | | |
| Yes | 191 | 76.6 (74.2–79.2) | 13.8 | 2.1 | 226 | 72.6 (76.5–78.2) | 15.3 | 15.4 |
| No | 1181 | 78.5 (77.7–79.3) | 13.2 | NS | 1160 | 77.4 (76.5–78.3) | 14.3 | *** |
| FAS II | | | | | | | | |
| Low | 196 | 77.0 (74.9–79.0) | 13.2 | 1.2 | 197 | 72.6 (69.9–75.2) | 17.1 | 8.1 |
| Middle | 759 | 78.1 (77.1–79.1) | 13.3 | NS | 762 | 77.2 (76.1–78.2) | 13.8 | *** |
| High | 417 | 79.4 (77.9–80.8) | 13.6 | | 427 | 78.5 (77.7–79.3) | 13.2 | |
| Smoking status | | | | | | | | |
| Current | 189 | 77.6 (75.4–79.8) | 13.4 | 1.5 | 184 | 73.1 (70.7–75.6) | 14.9 | 10.3 |
| Former | 96 | 76.6 (73.2–80.0) | 13.9 | NS | 89 | 73.0 (68.6–77.1) | 15.2 | *** |
| Never | 1087 | 77.6 (75.4–79.8) | 13.1 | | 1113 | 77.7 (76.9–78.6) | 13.7 | |
| Physical activity (METs/week) | | | | | | | | |
| Low | 49 | 77.2 (76.1–78.4) | 14.9 | 4.1 | 128 | 71.9 (68.4–75.6) | 14.9 | 14.4 |
| Moderate | 556 | 79.4 (78.5–80.5) | 13.1 | * | 739 | 75.8 (74.7–76.9) | 14.1 | *** |
| Vigorous | 767 | 79.7 (74.4–84.9) | 13.2 | | 519 | 79.4 (78.1–80.7) | 13.9 | |
| Sexual debut | | | | | | | | |
| Yes | 255 | 78.9 (76.9–80.9) | 13.6 | 0.2 | 181 | 74.9 (72.5–77.5) | 14.9 | 2.6 |
| Not yet | 1117 | 78.6 (77.7–79.4) | 13.3 | NS | 1205 | 77.1 (76.2–77.9) | 14.3 | NS |
| BMI (kg/m$^2$) | | | | | | | | |
| Obese and Overweight | 243 | 77.8 (75.9–79.7) | 13.7 | 2.4 | 173 | 73.4 (70.7–76.2) | 13.1 | 4.6 |
| Underweight | 112 | 76.2 (73.5–78.8) | 13.2 | NS | 167 | 77.1 (74.9–79.3) | 14.3 | * |
| Normal | 1017 | 79.1 (78.1–79.9) | 13.3 | | 1046 | 77.3 (76.4–78.2) | 14.3 | |
| Self-rated health | | | | | | | | |
| Poor | 22 | 70.1 (62.7–77.7) | 12.3 | 34.5 | 48 | 64.9 (59.1–70.8) | 16.3 | 39.7 |
| Fair | 60 | 67.7 (63.4–72.1) | 15.1 | *** | 112 | 69.2 (66.1–72.3) | 15.3 | *** |
| Good | 332 | 73.5 (71.9–75.1) | 13.5 | | 473 | 73.7 (72.3–75.1) | 14.7 | |
| Very good | 630 | 79.9 (78.9–80.9) | 11.8 | | 574 | 79.3 (78.2–80.4) | 12.4 | |
| Excellent | 328 | 83.3 (81.9–84.7) | 12.1 | | 179 | 85.2 (83.4–86.9) | 11.1 | |
| Youth disability | | | | | | | | |
| Yes | 342 | 72.5 (70.7–74.3) | 14.2 | 89.1 | 406 | 70.4 (68.6–72.2) | 13.9 | 104 |
| No | 1030 | 80.6 (79.9–81.4) | 11.7 | *** | 980 | 79.5 (78.6–80.3) | 12.5 | *** |

Significantly different * at $p<0.05$; ** at $p<0.01$ *** at $p<0.001$

tia of the three dimensions, and squared correlations with each dimension labeled cosine[2]. These statistics, available at the author, revealed high quality of solution.

In summary, associations were found between both objective (based on parental reports) and subjective (based on adolescents' reports) measures of family's SES as well as various daily lifestyle health behaviours. These associations conformed to the pattern: low SES was associated with less physical activity, cigarette smoking and early initiation of sexual activity.

*Socioeconomic and lifestyle predictors of the quality of life* Results of the one-way ANOVA conducted to compare the influence of selected socioeconomic and lifestyle behaviour factors on the quality of life are presented in Table 2. The table contains the mean QoL total perceptual score (QoLTPS), 95% confidence interval, and standard deviation sorted by the level of SES and lifestyle factors, and by gender group of boys and girls.

There is evidence of gender disparities in QoL in response to SES and lifestyle variables. In boys, the mean QoL total perceptual score (QoLTPS) for all but physical activity, self-rated health and youth disability did not show statistically significant differences among levels of SES and lifestyle variables. Unlike boys, girls did show such differences with regard to age group, financial strain, adolescents' FAS, physical activity, BMI, self-rated health and youth disability. Moreover, a gradient across SES and lifestyle behaviour groups was observed for QoL.

Physical activity was the lifestyle factor significantly contributing to adolescents' QoL ($F$: 2, 1,383=14.4 $p<0.001$ for boys as compared to $F$: 2, 1,383=14.4 $p<0.001$ for girls). Results of the Tukey's test revealed that boys performing physical activity at moderate to vigorous levels of intensity were likely to perceive their well-being significantly better than their peers performing physical activity at low level of intensity (QoLTPS means 79.7 and 79.4 as compared to 77.2). For girls, all levels of intensity in physical activity were likely to differentiate significantly their well-being (the mean QoLTPS for groups of girls performing physical activity at low, moderate and vigorous levels of intensity were 71.9, 75.8, and 79.4, respectively).

In boys and girls alike, all levels of the self-rated health factor significantly affected QoL ($F$: 4, 1,367=34.5 $p<0.001$ and $F$: 4, 1,386=39.7 $p<0.001$ for boys and girls respectively). There was a positive linear relationship between increasing perceived health and the proportion of boys and girls that better evaluated their QoL. The lowest mean value of QoLTPS was found for those who perceived their health as poor/fair (70.1;67.7 in boys as compared to and 64.9; 69.2 in girls). The highest mean value of QoLTPS was found in those who perceived their health very good (79.9 boys and 79.3 girls) and excellent (83.3 in boys and 85.2 in girls). This positive SES and lifestyle gradient in QoL was manifested in all variables but the age group where younger girls were likely to perceive QoL better than their older counterparts.

Girls living in better-off families were likely to perceive QoL better than their peers living in families under economic stress. This was true for both objective and subjective indicators of family affluence i.e. financial strain (FS) and FAS II (the mean QoLTPS for adolescent girls from families of economic burden was 72.6 as compared to a mean of 78.5 for families of high affluence). The overweight/obesity

Table 3. Univariate analyses of covariance (ANCOVAs) of Quality of Life Total Perceptual Score with selected social, cultural and health variables as predictors, and age as covariate

| | One main effect | | Covariate | |
|---|---|---|---|---|
| | | | Age | |
| Gender | $F$=9.9 | $p$<0.01 | $F$=2.5 | NS |
| Resident area | $F$=0.8 | NS | $F$=2.1 | NS |
| Mother education | $F$=2.1 | NS | $F$=2.9 | NS |
| Financial strain | $F$=16.6 | $p$<0.001 | $F$=2.1 | NS |
| FAS II | $F$=8.7 | $p$<0.001 | $F$=2.7 | NS |
| Physical activity | $F$=16.7 | $p$<0.001 | $F$=0.1 | NS |
| Smoking status | $F$=8.7 | $p$<0.001 | $F$=0.1 | NS |
| Sexual debut | $F$=0.1 | NS | $F$=2.1 | NS |
| BMI | $F$=6.2 | $p$<0.05 | $F$=1.8 | NS |
| Self rated health | $F$=72.9 | $p$<0.001 | $F$=0.2 | NS |
| Youth disability | $F$=195.1 | $p$<0.001 | $F$=1.5 | NS |

status was associated with lower perception of QoL as compared to girls of normal weight status ($F$: 2, 1,386=39.7 $p$<0.001; mean QoLTPS was 77.3 for normal weight as compared to 73.4 for overweight/obese). Another interesting result is that girls who never smoked were likely to perceive QoL better than their smoking counterparts (mean QoLTPS was 77.7 for never as compared to 73.1 for current smokers).

In the next step of our analysis, we performed ANCOVA models to estimate which of the single SES and lifestyle factors were predictive to adolescents' QoL. All estimations were done under control on chronological age (covariate variable).

Table 4. Multivariate analyses of covariance (ANCOVA) of Quality of Life Total Perceptual Score with selected social, cultural and health variables as predictors, and age as covariate

| Two main effects | | | Covariate | | Interaction effect | | |
|---|---|---|---|---|---|---|---|
| | | | Age | | | | |
| Gender and | $F$=8.6 | $p$<0.01 | | | | Gender × FS | |
| Finan. strain | $F$=13.6 | $p$<0.001 | $F$=2.1 | NS | $F$=6.1 | $F$=2.4 | NS |
| Gender and | $F$=12.2 | $p$<0.001 | | | | Gender × FAS II | |
| FAS II | $F$=8.8 | $p$<0.001 | $F$=2.5 | NS | $F$=6.6 | $F$=1.7 | NS |
| Gender and | $F$=11.4 | $p$<0.001 | | | | Gender × PhA | |
| Phys. activity | $F$=12.4 | $p$<0.001 | $F$=0.1 | NS | $F$=7.9 | $F$=4.2 | $P$<0.05 |
| Gender and | $F$=0.2 | ns | | | | Gender × SRH | |
| SRHealth | $F$=66.7 | $p$<0.001 | $F$=0.2 | NS | $F$=1.9 | $F$=1.1 | NS |
| Gender and | $F$=10.4 | $p$<0.01 | | | | Gender × Smoking | |
| Smoking | $F$=8.3 | $p$<0.001 | $F$=0.4 | NS | $F$=5.3 | $F$=2.1 | NS |

| Three main effects | | | Age | | | Interaction effect | |
|---|---|---|---|---|---|---|---|
| | | | | | | Gender × FAS II | |
| Gender and | $F=9.0$ | $p<0.01$ | | | | $F=0.2$ | NS |
| FAS II and | $F=3.7$ | $p<0.05$ | | | | Gender × PhA | |
| Phys. activity | $F=13.6$ | $p<0.001$ | $F=0.1$ | NS | $F=8.3$ | $F=3.7$ | $p<0.05$ |
| | | | | | | FAS II × PhA | |
| | | | | | | $F=0.9$ | NS |
| | | | | | | Gender × FAS II × PhA | |
| | | | | | | $F=1.5$ | NS |
| | | | | | | Gender × FAS II | |
| Gender and | $F=17.1$ | $p<0.001$ | | | | $F=3.8$ | $p<0.05$ |
| FAS II and | $F=0.9$ | ns | | | | Gender × Smoking | |
| Smoking | $F=7.2$ | $p<0.001$ | $F=0.4$ | NS | $F=4.9$ | $F=6.4$ | $p<0.001$ |
| | | | | | | FAS II × Smoking | |
| | | | | | | $F=2.4$ | NS |
| | | | | | | Gender × FAS II × Smoking | |
| | | | | | | $F=4.4$ | $p<0.01$ |

Univariate analysis (Table 3) revealed that of all factors in question, the following ones appeared to be predictive to QoL: gender ($F=9.9$ $p<0.01$), economic factor (financial strain and FASII; $F=16.6$ $p<0.001$ and $F=8.7$ $p<0.001$, respectively), lifestyle behaviours such as habitual physical activity ($F=16.7$ $p<0.001$) and smoking status ($F=8.7$ $p<0.001$), as well as health indicators such as weight status, self-rated health and disability condition ($F=6.2$ $p<0.05$; $F=72.9$ $p<0.001$, and $F=195.1$ $p<0.001$, respectively). Of all factors taken to analysis, health disability appeared to be the most predictive to adolescents' quality of life, followed by self-rated health, family economic burden and physical activity.

None of the univariate models revealed significant contribution of chronological age to variation in adolescent QoL. That is each factor significantly affected adolescent QoL irrespective of their chronological age.

It has been known however that significant effect of selected factor found in uni-variate approach often disappears when it is involved in combination with other factors. In the next step of our analysis, multivariate ANCOVA models were created involving two and three factors and tested for their main effect on adolescent QoL. The findings (Table 4) revealed that gender appeared to be significant predictor of adolescents' QoL in combination with all other factors studied (i.e. financial strain, FASII, physical activity and smoking status) but the self-rated health. In the model involving gender and physical activity, the interaction of these two factors significantly contributed to variation in QoL. Other models did not reveal this rule.

Moreover, gender remained significantly predictive factor for QoL in combination with two other variables. The three-variate models revealed three main factors, each including gender, economic and health behaviour factors: (1) gender, FAS II and physical activity, and (2) gender in combination with FAS II and smoking status.

## Discussion

Results of parental and adolescent reports on socioeconomic status along with a number of health-related behaviours, have outlined co-occurrence of these two sets of variables. The urbanization factor and level of maternal education appeared to be associated with economic status of family and adolescents' risk-for-health behaviours. Rural families, typically associated with low level of maternal educational attainment, were likely to provide greater opportunities for financial disadvantages and taking unhealthy behaviours than their better educated counterparts living in urban areas. Urban adolescents from middle affluent families, where mother had completed at least 12 years of schooling, appeared to perform physical activity in moderate to vigorous intensity. They were likely to be more active physically than their peers coming from less wealth families. Among adolescents, current smokers were likely to initiate sexual activity at significantly younger age than their never smoked counterparts.

It has been widely accepted that most behaviours are socially patterned and often occur together. Many people who follow health-promoting dietary practices also tend to be physically active. Those who drink also use tobacco and tend to be substance abuse. People who are poor, have low levels of education, or are socially isolated are more likely to engage in a wide array of risk-related behaviours and less likely to engage in health-promoting ones. The direction of associations between SES and lifestyle choices yielded in the present study, revealed a social status gradient in health-enhancing behaviour: low SES was likely to be associated with risk-for-health behaviours such as less physical activity, cigarette smoking and early initiation of sexual activity.

Literature review shows that findings on the relationship between SES and various measures of daily lifestyle health behaviour during the period of adolescence have been very heterogeneous [Hanson and Chen 2007]. In general, they can conform to three patterns: (1) patterns found in adulthood suggesting that lower SES is associated with poorer health behaviours [Bielicki and Welon 1982; Williams 1990; Adler et al. 1994; Mazur 2010], (2) reversed associations suggesting that high SES is related to greater negative health behaviour [Luthar and Becker 2002; Luthar and Latendresse 2005], and (3) null/weak associations suggesting non-significant relationships between SES and health behaviours [West and Sweating 2004; Friedstad and Klepp 2006]. Present study provided support for consistently documented social class gradients in Polish child and adolescent health behaviours which have been listed above in the first place [Mazur 2010].

With knowledge about the structure of contextual variables i.e. SES and lifestyle variables as a starting point, the present study focused on the perceived psychosocial well-being in a young sample (aged 13–18) of the general Polish population, providing norms and investigating the influence of SES and lifestyle variables.

In general, the majority of adolescent boys and girls in the present study reported positive global QoL. This is in accordance with the results obtained in other populations [Huebner et al. 2005; Funk et al. 2006]. The mean YQOL-R total per-

ceptual scores for the group of boys were 78.6 compared to a mean of 76.8 for the group of girls. This difference was statistically significant ($F=9.5$ $P<0.01$) and gender accounted for approximately 0.7% of the variation in total QoL scores. Further analyses, using mixed models ANOVA/ANCOVA, were conducted in order to identify single relevant factors.

Results of the one-way ANOVA provided different picture of the influence of contextual variables on the global quality of life for boys as compared to girls. Boys' quality of life was significantly differentiated by self-rated health status, health disability and intensity of physical activity. Girls' quality of life was differentiated by age, family wealth, health behaviours such as intensity of physical activity, weight status and smoking habit, as well as self-rated health status and health disability. A review of the literature about age and gender effects on QoL or on life satisfaction indicates inconsistent findings [Michel et al. 2009]. Most studies, including the present one, report a poorer QoL in healthy teenage girls compared to boys [Woynarowska et al. 2002; Bisegger et al. 2005]. The same tendency has been observed in adolescents with chronic conditions such as diabetes [Graue et al. 2003; Wagner et al. 2004], and cystic fibrosis [Arrington-Sanders et al. 2006]. In contrast to those data, the study by Huebner et al. [2004] has shown no significant gender or grade in school differences of life satisfaction. Inconsistencies between the studies could be due to different methods of measuring quality of life (Huebner et al. has administered the Brief Multidimensional Students' Life Satisfaction Scale) and due to the different developmental stage/age of the study population. Different findings have been obtained for mixed group of pre- and post-pubertal individuals as compared to homogenous being either before or after puberty. Goldbeck et al. [2007] has demonstrated that developmentally determined changes in life satisfaction are more likely in the phase between 11 and 16 years of age. This age span can be assumed as the one in which the most significant growth and developmental changes occur. In the present study, only girls revealed significant age effect with decreasing quality of life. Statistically significant mean differences of YQOL-R total perceptual scores for the group of younger girls, aged 13–15, and students of junior secondary school (gymnasium) were 78.2 compared to a mean of 75.7 for the older, aged 16–18, students of senior secondary school ($F=8.3$ $p<0.05$).

Urbanization (the concentration of people and activities into areas classified as urban) is one of the most important demographic shifts worldwide during the past century and is set to continue well into the 21st century [UNEP 2000]. Urban environments have been linked to a range of positive and negative implications for human health issues. Cities offer the opportunities for better employment, education, health care, and culture. However, rapid and often unplanned urban growth is often associated with environmental degradation, pollution and industrial waste, increased motor vehicle traffic, lack of urban services, and stress associated with poverty and unemployment, among others [Moore et al. 2003; Kaczmarek and Skrzypczak this volume]. There is growing evidence that urban living negatively affect health. In the present study, living in urban or rural areas appeared not to differentiate significantly adolescents' QoL.

A series of univariate analyses of covariance (ANCOVA models) using the scores of YQOLTPS as dependent variable, and gender, urbanization, maternal education, family economic status, physical activity, smoking habit, initiation of sexual activity, body mass index (BMI), self-rated health and health disability category as fixed factors, and controlling for chronological age showed that from all contextual variables included in analyses, those linked to health (health disability and self-rated health), economic situation of one's family (evaluated objectively and subjectively) and physical activity were most significant to adolescents' QoL. Students with disabilities and self-rated poor health reported a significantly lower QoL than those without and with self-rated excellent health. This finding is in agreement with other studies of the relationships between adolescents with perceived disabilities and perceived health [Edwards et al 2003; Mazur 2010].

Students living in families with economic burden (evaluated objectively by parents and subjectively by students themselves) reported a significantly lower QoL than their more affluent counterparts. At the same time, the residence place and maternal education level appeared not to be significantly associated with adolescent QoL. This finding implies that indicators of economic status (material well-being) are likely to play more important role in adolescents' assessment of their QoL than traditional indicators of SES (mother educational attainment) and corroborates well with some previous studies of the relationship between individual and family factors and adolescent well-being [Conger et al. 2000; Ben-Zur 2003; Mazur 2010].

The results of present study showed that active students have a better QoL irrespective their gender and age than their counterparts performing sedentary lifestyle. There are numerous studies supporting this finding [Gillison et al. 2006; Humbert et al. 2006; Sánchez-López 2009].

A series of multivariate analyses of ANCOVA with chronological age as covariate provide support toward the contention that gender, economic and lifestyle health behaviour factors are main predictors of adolescent QoL.

Hence, there are several limitations to this study that must be taken into account. First, the cross-sectional nature of this analysis does not warrant conclusive statements about the causal nature of the relationships. This requires further investigation. Second, self-report lifestyle health behaviour measures can result in underestimates or overestimates of particular variables. The use of objective assessments of these variables would corroborate and add further validity to our findings.

The strengths of this study lie in the setting and design. The study was a population-based one with an age- and sex-matched. Validated measures were used to assess the psychosocial and family factors of the study group as well as somatometrics assessing physical growth and development.

## Conclusion

The hypothesis that adolescents of low socioeconomic status and unhealthy lifestyle behaviours would report significantly lower perceived QoL scores than their

better-off and healthy lifestyle behaviour counterparts was supported. Gender, economic and lifestyle behaviour factors were identified as main predictors of adolescent QoL.

Quality of life in general population of adolescents has been scarcely documented. Our findings provide useful information on variation in adolescent QoL and its main contextual and intraindividual predictors. This knowledge, essential to assess how adolescents perceive their own situation, may be used for further theoretical development. It also can be translated to practice and used for further consideration of intervention strategy in the rising tide of child and adolescent physical and mental health.

A further challenge lies in explaining the mechanism of socieoeconomic and lifestyle health constraints which significantly affect adolescent QoL.

## References

Aaron D.J., Dearwater S.R., Anderson R., Olsen T., Kriska A.M., Laporte R.E.: Physical activity and the initiation of high-risk health behaviors in adolescents. *Med Sci Sports Exerc* 1995: 27: 1639–1645.

Adler N.E., Boyce T., Chesney M.A., Cohen S., Folkman S., Kahn, R.L., Syme S.L.: Socioeconomic status and health: The challenge of the gradient. *Am Psychologist* 1994: 49: 15–24.

Ainsworth B.E., Haskell W.L., Leon A.S., Jacobs Jr D.R., Montoye H.J., Sallis J.F., Paffenbarger Jr R.S.: Compendium of physical activities: classification of energy costs of human physical activities. *Med Sci Sports Exerc* 1993: 25: 71–80.

Ainsworth B.E., Haskell W.L., Whitt M.C., Irwin M.L., Swartz A.M., Scott J.S., O'Brien W.L., Bassett Jr D.R., Shmitz K.H., Emplaincourt P.O., Jacobs Jr D.R., Leon A.S.: Compendium of Physical Activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc* 2000: 32(9 Suppl): S498-S516.

Ainsworth B.E., Haskell W.L., Herrmann S.D., Meckes N., Bassett Jr D.R., Tudor-Locke C., Greer J.L., Vezina J., Whitt-Glover M.C., Leon A.S.: Compendium of Physical Activities: a second update of codes and MET values. *Med Sci Sports Exerc* 2011: 43(8): 1575–1581.

Arrington-Sanders R., Yi M.S,, Tsevat J., Wilmott R.W., Mrus J.M., Britto M.T.: Gender differences in health-related quality of life of adolescents with cystic fibrosis. *Health Qual Life Outcomes* 2006: 24(4): 5–8.

Armstrong D., Caldwell D.: Origins of the Concept of Quality of Life in Health Care: a Rhetorical Solution to a Political Problem. *Soc Theory Health* 2004: 2(4): 361–371.

Bańka A.: *Psychologia jakości życia*. Stowarzyszenie Psychologia i Architektura, Poznań 2005.

Ben-Zur H.: Happy adolescents: The link between subjective well-being, internal resources, and parental factors *J Youth Adolescence* 2003: 32(2): 67–79.

Bielicki T., Welon Z.: Growth data as indicators of social inequalities: the case of Poland. *Yearbook Phys Anthropol* 1982: 25: 153–167.

Bisegger C., Cloetta, B. von Rueden U., Abel T., Ravens-Sieberer U.: European Kidscreen Group.: Health-related quality of life: gender differences in childhood and adolescence. *Soz Praventivmed*, 2005: 50: 281–291.

Bloom D.E., Craig P.H., Malaney P.N.: *The Quality of Life in Rural Asia*. Hong Kong: Oxford University Press 2001.

Bradburn N.M.: *The structure of psychological well-being*. 1969. Chicago, Ill.: Aldine. Quoted in: Campbell A.: Subjective measures of well-being. *Am Psychologist* 1976: 2: 119.

Campbell A.: Subjective measures of well-being. *Am Psychologist* 1976: 2: 117–124.
Campbell A., Converse P.E.: (Eds.) *The Human Meaning of Social Change*. 1972. New York: Russel Sage Foundation. Quoted in: Campbell A.: Subjective measures of well-being. *Am Psychologist* 1976: 2: 120.
Cantril H.*: The Pattern of Human Concerns*. 1965. New Brunswick, N.J.: Rutgers University Press. Cited after: Campbell A.: Subjective measures of well-being. *Am Psychologist* 1976: 2: 119.
Cole T.J., Bellizzi M.C., Flegal K.M., Dietz W.H.: Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 2000: 320: 1240–1243.
Cole T.J., Flegal K.M., Nicholls D., Jackson A.A.: Body mass index cut offs to define thinness in children and adolescents: international survey. *BMJ* 2007: 335: 194–201.
Cohen S., Alper C.M., Doyle W.J., Treanor J.J., Turner R.B.: Positive emotional style predicts resistance to illness after experimental exposure to rhinovirus or Influenza A virus. *Psychosom Med* 2006: 68: 809–815.
Conger K.J., Rueter M.A., Conger R.D.: The role of economic pressure in the lives of parents and their adolescents: The family stress model. In: L.J. Crockett and R.K. Silbereisen (Eds.), *Negotiating adolescence in times of social change* New York: Cambridge University Press. 2000: 201–233.
Constanza R., Fisher B., Ali S., Beer C., Bond L. et al.: An integrative approach to quality of life measurements, research, and policy. S.A.P.I.E.N.S. [Online] 2008: 1(1). Online since 19 December 2008. URL: http: //sapiens.revues.org/169 Accessed February 22, 2011.
Cramer J.A., Spilker B.: *Quality of Life and Pharmacoeconomics: An Introduction*. Philadelphia, PA: Lippincott-Raven Publishers, 1998.
Currie C.E., Elton R.A., Todd J., Platt S.: Indicators of socioeconomic status for adolescents: the WHO Health Behaviour in School-aged Children Survey. *Health Educ Res* 1997: 12: 385–397.
Czapiński J., Panek T.: (Eds.) Diagnoza społeczna 2011. *Warunki i jakość życia Polaków*. Warszawa. Rada Monitoringu Społecznego: Available at http://www.diagnoza.com.pl. Accessed March 14, 2011.
Day H., Jankey S.G.: Lessons from the literature. Towards a holistic model of quality of life. In: R. Renwick, I. Brown, and M. Nagler (Eds.) *Quality of Life in Health Promotion and Rehabilitation. Conceptual Approaches, Issues and Applications*. Thousand Oaks: Sage 1996: 39–50.
Daly H.E., Cobb J.B.: *For the Common Good*, 1989. Boston: Beacon Press.
Davis E., Waters E., Mackinnon A., Reddihough D., Graham H.K., Mehmet-Radji O., Boyd R.: Paediatric quality of life instruments: a review of the impact of the conceptual framework on outcomes. *Dev Med Child Neurol* 2006: 48: 311–318.
Edwards T.C., Huebner C.E., Connell F.A., Patrick D.L.: Adolescent quality of life, Part I: conceptual and measurement model. *J Adolescence* 2002: 25: 275–286.
Edwards T.C., Patrick D.L., Topolski T.D.: Quality of life of adolescents with perceived disabilities. *J Pediatr Psychol* 2003: 28(4): 233–241.
Farquhar M.: Definitions of quality of life: a taxonomy. *J Adv Nurs* 1995: 22(3): 502–509.
Ferries A.F.: The quality of life concept in sociology. *Am Sociol* 2004: 35(3): 37–51.
Friestad Ch., Knut-Inge Klepp K-I.: Socioeconomic status and health behaviour
patterns through adolescence: Results from a prospective cohort study in Norway. *Eur J Public Health* 2006: 16(1): 41–47.
Funk B.A., Huebner E.S., Valois R.F.: Reliability and validity of a brief life satisfaction scale with a high school sample, *J Happiness Stud* 2006: 7(1): 41–54.
Gillison F.B., Standage M., Skevington S.M.: Relationships among adolescents' weight perceptions, exercise goals, exercise motivation, quality of life and leisure-time exercise behaviour: a self-determination theory approach. *Health Edu Res* 2006: 21(6): 836–847.

Goldbeck L., Schmitz T.G., Besier T., Herschbach P., Henrich G.: Life satisfcation decreases during adolescence. *Qual Life Res* 2007: 16: 969–979.
Graue M., Wentzel-Larsen T., Hanestad B.R., Båtsvi B., Søvik O.: Measuring self-reported, health-related, quality of life in adolescents with type 1 diabetes using both generic and disease-specific instruments. *Acta Paediatrica* 2003: 92: 1190–1196.
Guyatt G.H., Kirshner B., Jaeschke R.: Measuring health status: what are the necessary measurements properties? *J Clin Epidemiol* 1992: 45: 1347–1351.
Hansen M.D., Chen E.: Socioeconomic status and health behaviors in adolescence: a review of the literature. *J Behav Med* 2007: 30: 263–285.
Holmes S.: Assessing the quality of life – reality or impossible dream? A discussion paper. Int *J Nurs Stud* 2005: 42(4): 493–501.
Huebner E.S., Suldo S., Valois R.F., Drane J.W., Zullig K.: Brief multidimensional students' life satisfaction scale: sex, race, and grade effects for a high school sample. *Psych Rep* 2004: 94: 351–356.
Huebner E.S., Valois R.F., Paxton R.J., Drane J.W.: Middle school student's perceptions of quality of life. *J Happiness Stud* 2005: 6(1): 15–24.
Humbert M.L., Chad K.E., Spink L.S., Muhajarine N., Anderson K.D., Bruner M.W., Girolami T.M., Odnokon P., Gryba C.R.: Factors that influence physical activity Participation among high- and low-SES youth. *Qual Health Res* 2006: 16(4): 467–483.
Kaczmarek M.: Koncepcja i pomiar jakości życia związanej ze zdrowiem człowieka. In: M. Kaczmarek and A. Szwed (Eds.) *Między antropologią a medycyną Koncepcje teoretyczne i implikacje praktyczne*. Poznań: Wydawnictwo Naukowe UAM. 2009: 25–49.
Kamerschen D.R., McKenzie R.B., Nardinelli C.: *Ekonomia*. 4th ed. 1999. Pelpin: Wydawnictwo "Bernardinum".
Knussmann R.: Somatometrie. In: *Anthropologie* (Hrsg. R. Knussmann). Stuttgart: Fischer Verlag. 1988: 232–285.
Langlois A., Anderson D.E.: Resolving the Quality of Life/Well-being Puzzle: Toward a New Model. *Can J Reg Science/Rev canad sciences region* 2002: XXV(3): 501–512.
Luthar S.S., Becker B.E.: Privileged but pressured? A study of affluent youth. *Child Dev* 2002: 73: 1593–1610.
Luthar S.S., Latendresse S.J.: Children of the affluent: Challenges to well-being. *Psychosom Med* 2005: 14: 49–53.
Mazur J.: *Społeczne nierówności w zdrowiu subiektywnym młodzieży szkolnej w Polsce na tle Unii Europejskiej. Wybrane aspekty metodologiczne ilustrowane wynikami międzynarodowych badań*. Warszawa 2010.
Meeberg G.: Quality of life: a concept analysis. *Aust J Adv Nurs* 1993: 18(4): 32–38.
Michel G., Bisegger C., Fuhr D.C., Abel T., The KIDSCREEN group: Age and gender differences in health-related quality of life of children and adolescents in Europe: a multilevel analysis. *Qual Life Res* 2009: 18: 1147–1157.
Mini A.M., Heron M., Murphy S.L., Kochanek K.D.: Deaths: Final Data for 2004. Health E-stats, National Center for Health Statistics, Hyattsville, MD 2006. Available at www.cdc.gov/nchs/data/databriefs/db26.htm. Accessed March 23, 2011.
Moore M., Gould P., Keary B.S.: Global urbanization and impact on health. *Int J Hyg Environ Health* 2003: 206(4–5): 269–78.
Nussbaum M.C., Sen A.: (Eds) *The Quality of Life*. Oxford, UK, Oxford University Press 1993.
Patrick D.L., Bergner M.: Measurement of health status in the 1990s. *Ann Rev Public Health* 1990: 11: 165–83.
Plackett R.L.: The old statistical account, *J Roy Stat Soc*, A, 1986: 149: 247–251.
*Preambule to the Constitution of the World Health Organization as adopted by the International Health Conference*, New York, 19–22 June, 1946: signed on 22 July 1946 by the represen-

tatives of 61 States (Official Records of the World Health Organization, no. 2, p. 100) and entered into force on 7 April 1948.
Raeburn J.M., Rootman I.: Quality of life and health promotion. In: W.R. Renwick, I. Brown and M. Nagler (Eds.) *Quality of Life in Health Promotion and Rehabilitation: Conceptual Approaches, Issues and Applications*. 1996: 14–25. Thousand Oaks Thousand Oaks, residential city (1990 pop. 104,352, Ventura co., S Calif., in a farm area: inc. 1964. Avocados, citrus, wegetables, strawberries, and nursery products are grown.
Raphael D.: Defining quality of life: eleven debates concerning its measurement. In: R. Renwick, I. Brown, and M. Nagler (Eds.) *Quality of Life in Health Promotion and Rehabilitation. Conceptual Approaches, Issues and Applications*. 1996. Thousand Oaks: Sage.
Rapley M.: *Quality of Life Research – a Critical Introduction*. 2003. Lonodn: SAGE Publication.
Rejeski W.J., Mihalko S.L.: Physical activity and quality of life in older adults. *J Gerontol: Series A: Biological Sciences & Medical Sciences* 2001: 56 (11): 23–35.
Romney D.M.: A structural analysis of health related quality of life dimensions. *Hum Relat* 2002: 45: 165–176.
Sánchez-López M., Salcedo-Aguilar F., Solera-Martínez M., Moya-Martínez P., Notario-Pacheco B., Martínez-Vizcaíno V.: Physical activity and quality of life in schoolchildren aged 11–13 years of Cuenca, Spain. *Scand J Med Sci Sports* 2009: 19(6): 879–884.
Schalock R.L.: The concept of quality of life: what we know and do not know. *J Int Dis Res* 2004: 48(3): 203–216.
Schipper H., Clinch J., Powell V.: Quality of life studies: definitions and conceptual issues. *Arthritis Rheum* 1988: 31: 315–24.
Schuessler K.F., Fisher G.A.: Quality of fife research and sociology. *Ann Rev Sociology* 1985: 11: 129–149.
Seligman M.E.P.: Positive health. *Appl Psych IAAP* 2008: 57: 3–18.
Spilker B., Molinek F.R., Johnston K.A., Simpson R.L., Tilson HH, Quality of life bibliography and indexes. Med. Care, 1990: 28 (suppl.): D1-D78.
Stenner P.H.D., Cooper D., Skevington S.M.: Putting the Q into quality of life : the identification of subjective constructions of health-related quality of life using Q methodology. *Soc Sci Med* 2003: 57: 2161–2172.
Taillefer M.C., Dupuis G., Roberge, M-A., Lemay S.: Health-Related Quality of Life
Models: systematic review of the literature. *Soc Ind Res* 2003: 64: 293–323.
Testa M.A.: Assessment of Quality-of-Life Outcomes. *NEJ Med* 1996: 334(13): 835–840.
Topolski T.D., Edwards T.C., Patrick D.L.: *Users's manual and interpretation guide for the Youth Quality of Life (YQOL) Intsruments*. Seattle, WA: University of Washington, Dept. of Health Services. 2002.
Townsend P.: Deprivation. *J Soc Policy* 1987: 16: 125–146.
UNEP (2000). The urban environment: facts and figures. *UNEP Industry and Environment*, 2000: 23(2): 4–11. United Nations.
Wagner J.A., Abbott G., Lett S.: Age related differences in individual quality of life domains in youth with type I diabetes. *Health Qual Life Outcomes* 2004: 2: 54–59.
Williams D.R.: Socioeconomic differentials in health: A review and redirection. *Soc Psych Quart* 1990: 32: 81–89.
West P., Sweeting H.: Evidence on equalization in health in youth from the West of Scotland. *Soc Sci Med* 2004: 59: 13–27.
Woynarowska B., Tabak I., Mazur J.: Self-reported health and life satisfaction in school-aged children in Poland and other countries in 2002. *Med Wieku Rozwoj* 2002: 8: 535–550.

Maria Kaczmarek, Magdalena Durda

# Variation in the body image perceptions of adolescent females and males and underlying social and cultural settings

**Abstract:** Satisfaction with physical appearance is crucial for the quality of life and well-being of adolescents. Dissatisfaction with body image may increase risks for body distortions, depression, and eating disorders. The aim of the study was to assess variation in (dis)satisfaction with body image among adolescent females and males in relation to social and cultural factors. The studied sample comprised 1552 boys and 1578 girls aged 13–18, participants of the ADOPOLNOR project. Perception of body size was evaluated using figural ratings method and controlled to BMI. Rate of global dissatisfaction with body image was correlated to age, gender, family socio-economic status and lifestyle behaviour of young people. Results of the logistic regression analysis revealed that gender and BMI were two variables most significantly associated with body image dissatisfaction ($p<0.001$). Socio-economic status of the family where young people were brought appeared no to be directly associated with body image dissatisfaction. However, associations of smoking, physical activity, and sexual activity with body dissatisfactions ($p<0.05$) showed that lifestyle behaviour may be an important predictor of poor body image in adolescence.

**Key words:** body image, adolescents, lifestyle, SES

*While the onset of puberty can vary by as much as six years, every adolescent wants to be right on the 50-yard line, right in the middle of the field. One is always too tall, too short, too thin, too fat, too hairy, too clear-skinned, too early, too late. Understandably, problems of self-image are rampant.*
Joan Lipsitz

*Easing the Transition From Child to Adult*
Education Week, May 16, 1984

# Introduction

## Defining body image

In Mirriam-Webster Medical Dictionary, the term body image is a noun and refers to a subjective picture of one's own physical appearance established both by self-observation and by noting the reactions of others [http://www.mirriam-webster.com].

American Heritage Dictionary defines body image as (i) the cerebral representation of all body sensation organized in the parietal cortex, and (ii) the subjective concept of one's physical appearance based on self-observation and the reactions of others [http:/dictionary.com].

Oxford Dictionary of Sport and Medicine presents the following definition of body image:

It is the perception, both conscious and unconscious, of one's own body and physical dimensions [www.answers.com].

Human physique, body appearance, physical attractiveness has interested philosophers and scientists for centuries. The ancient Greek philosophers believed that there is a fundamental relation between body and mind, beauty and positive qualities. People who are beautiful are also good, whereas, ugly people are usually attributed with negative qualities [Langlois et al. 2000].

Scientific interest in the issue of perception own body (body image) dates back to the beginning of the 20th century. This early period of body image research dealt with body experience induced by dysfunction or traumatic brain injuries and focused on explaining the pattern of the body (body schema), a hypothetical neurological mechanism that was responsible for changes in body posture and body movements performed [Pruzinsky, Cash 2004:4].

The founder of the body image concept is considered to Paul Schilder, Austrian neurologist and psychoanalyst, who first exceeded in his research outside the boundaries of neuropathology. In a book entitled *The Image and Appearance of the Human Body*, he introduced a biopsychological perspective of body image and the neurological, psychological and sociocultural components of this construct. Schilder defined body image as "the tridimensional image everyone has about himself, the picture of our own body which we form in our mind in its physiological, libidinous, and sociological aspects" [Schilder 1935/1950 reprinted 1999:11]. He described associations of body image with the postural and tactile impressions, localization, imperceptions of various kinds, synesthesia, apraxia, agnosia, the phantom, muscle-tone, and pain (the physiological basis of the image), narcissism, erogenic zones, neurasthenia, depresonalization, hypochondria, hysteria, and conversion (the libidinous aspect of body image), and curiosity, the expression of emotions, imitation, identification, beauty, and other social aspects of the body image. Schilder's insight into multifaceted nature of body image allowed him to foresee "most of the modern lines of research dealing with body experience" [Schilder 1935/1950 reprinted 1999; Fisher 1990:12].

An important place in the history of body image research takes Seymour Fisher who developed a theory of body image boundaries and empirically verified it among people with normal behaviour as well as with medical and psychiatric disorders. In his research, he focused on „barrier" and „penetration" boundary dimensions posited to reflect the strength of permeability of body boundaries. He expanded the understanding of body image with new terms such as "…the body image boundary, assignment of meaning to specific body areas, general body awareness, and distortion in body perception" [Fisher 1986 after: Cash, Smolak 2011:4].

The next important step in the study of body image was the work of Franklin Schontz. In his opus *Perceptual and Cognitive Aspects of Body Experience* [1969] and other works, he came out with body image as a multifaceted body experience (with seven functions and at least four levels of these functions), thus departing from the psychodynamic paradigm [Cash, Smolak 2011:4–5]. Schontz applied his two-dimensional (cognitive and perceptual) model of body experience to the study of physical disability and health psychology.

The nineties of the 20th century marked the rapid progress in studies on body image. Terminology increased quickly as body image research progresses. This led to a situation where there were 16 different terms used as synonyms for some facet of body image. Terms such as weight satisfaction, accuracy size perception, body satisfaction, appearance satisfaction, appearance evaluation, appearance orientation, physical attractiveness, body esteem, body concern, body dysmorphia, body schema, body percept, body distortion, body image, body image disturbance, and body image disorder, were used interchangeably causing difficulties in understanding the phenomenon of body image [Thompson et al. 1999:10].

Attempts were made to conceptualize body image through its different components. Brouwers distinguished three components: (i) physiological or the brain's ability to detect weight, shape, size, and form; (ii) conceptual, including formation of a mental picture of one's own body; and (iii) emotional, or perceived feelings about one's body weight, shape, and size [Brouvers 1990]. Altabe and Thompson conceptualized body image as a complex construct, considered to be, and measured as a multidimensional construct which consists of both psychological and physiological components. They listed four dimensions of body image: perceptual, emotional, mental and kinetic [Altabe, Thompson 1993].

Thompson and colleagues [1999] defined body image as "…a term that has come to represent the "internal" image or representation that we have of our physical appearance. It is to be contrasted with the "outer" image or an objective view of attractiveness (i.e., a rating made by a supposedly unbiased observer). Although commonly thought of as overlapping substantially, in fact one's inner view (body image) is only minimally correlated with actual ratings of attractiveness" [Thompson et al.1999:12].

Body image is considered of being a multidimensional construct. According to Cash and Pruzinsky, body image is „…a psychological construct of growing scientific and clinical interest. Having being diversely conceptualized throughout its long history, body image is currently regarded as multidimensional self-attitudes

toward one's body, particularly its appearance" [Cash, Pruzinsky 1990:18] but not exclusively one's physical appearance. Its predictors include internal biological and psychological factors as well as external cultural and societal ones [Cash, Pruzinsky 1990]. There are: the perception, attitude, cognition, behaviour, affect, fear of fatness, body distortion, body dissatisfaction, cognitive-behavioral investment, evaluation, preference for thinness, and restrictive eating indicated among many other dimensions of body image. No need to point out that understanding of body image is important both for theoretical considerations and practical application in the treatment of own body perception disorders. Frequently used concept of body image is a four-dimensional model including perceptual, cognitive, affective and behavioural dimensions [Cash, Smolak 2011]. The perceptual dimension of body image is defined as the accuracy of individuals' judgment of their size, shape, and weight relative to their actual proportions. The study of perceptual body image involves assessing the accuracy of body size estimations, either at the level of individual body parts or the body as a whole. The cognitive dimension relates to thoughts and beliefs concerning body shape and appearance. The affective dimension can be conceptualized as the feelings individuals have towards their bodies' appearance. The behavioural aspect of body image usually results from the remaining dimensions. The negative feelings associated with the perception of the body can lead to behavioural disturbances and vice versa, behavioural disturbances can cause problems in the affective and cognitive dimensions of body image.

As already said, the concept of body image has aroused great interest among both researchers and practitioners over the past few decades. Cash and Pruzinsky noted that there was an influential escalation of "body image" and "body (dis)satisfaction" citations in the psychological (PsycINFO) and medical (PubMed) databases from 100 (PsycINFO) and only very few in the PubMed in the 1951–1960, to 2,500 and 2,800 in PsycINFO in PubMed, respectively in the decade 1991–2000 [Cash, Pruzinsky 2004:9]. In the first decade of the 21st century, the number of body image publications in each database more than doubled relative to the 1990s [Cash, Smolak 2011:7].

Since 2004, the journal Body Image has published numerous original research articles on various topics related to body image. These results have been successfully applied to the design of prevention and intervention programmes, basically for people with body image distortions.

## Theoretical frameworks for viewing and investigating human body image issues

Conceptual approaches for understanding the origin and causal mechanisms underlying human appearance and body image effects include a set of wide-ranging perspectives such as biological (through genetic-environment interaction), neuroscientific, cognitive-behavioral, sociocultural, evolutionary, feminist (objectification theory), and positive psychology viewpoints.

Core assumptions and research tasks of the sociocultural, evolutionary, genetic and neuroscientific frameworks will be reviewed briefly.

The sociocultural perspective is an approach to understanding the ways societal factors influence the development and maintenance of body image across the lifespan [Tiggemann 2011; Jackson 2004]. The main premise of this approach is that social factors influence development of body image through culturally induced body appearance which emphasizes the desirability of physical attractiveness and beauty [Thompson et al. 1999]. Several theories have generated testable hypotheses on the role of physical attractiveness in human behaviour and its impact on how individuals are perceived by others and how they perceive themselves. These are the socialization (status generalization) and social expectancy theories, the implicit personality and social comparison theories [Jackson 2004:14]. Socialization/social expectancy theories argue that social standards of beauty exist within a particular culture. Such standards of body image values reinforce and model cultural ideals of beauty and body shape in stereotypically uniform ways [Field et al. 1999]. The body ideals for physical appearance are then transmitted via a variety of sociocultural pathways (among many others: appearance media exposure, appearance conversations with friends, and peer appearance criticism) and internalized by individuals resulting in development of differential behaviour and traits in attractive and unattractive targets (according to the implicit personality theory). The acceptance and adoption of societal ideals as goals for oneself can also proceed through social comparison or perceived pressure from the mass media. It is believed that the media perform an important role in shaping culture fashion and appearance standards [Jones et al. 2004]. Differential judgment and treatment eventually develop differential behaviour and self-view, either satisfaction or dissatisfaction with body image. In this way, satisfaction with body image will be a function of the extent to which individuals do or do not meet the ideal prescription [Tiggemann 2011:13].

Consistent with the sociocultural perspective is an example of the realm of body weight and shape. The Western culture idealizes thinness for females, the body shape, where the waist is slim and the hips are slightly wider and proportionate. This ideal body appearance is then transmitted by sociocultural channels, most notably by media, peers and family (parents, siblings) and incorporated by many women, especially adolescents. However, for some of girls and young women, it is virtually impossible to match up to this thin ideal and they become dissatisfied with their actual figure. The potentially negative consequences of the thin ideal include negative body image, low self-esteem, and psychological and physical disorders of life-threatening proportions which may eventually result in eating pathology among girls and young women.

Socioculturally transmitted body ideal for men is a mesomorphic and muscular V-shaped body with broad shoulders and a solid chest and a smaller lower body part. This ideal body is impossible for most men to achieve by healthy means resulting in body dissatisfaction or pursue of muscularity by unhealthy behaviours (taking steroids, supplements or any other).

Despite the said sociocultural pressure for men and women to adhere to body ideals in Western society, only some individuals develop body image problems. It

therefore has been hypothesized that susceptibility to problems with body image may be influenced by genetic factors. Twin studies in adolescent and young adult females, have suggested moderate to large genetic influences on a range of body image problems with heritability estimates 50% or higher [Klump et al. 2009; Wade et al. 2001].

Recent advances in integrating noninvasive functional magnetic resonance imaging (fMRI) with genetics, have enabled researchers to investigate the associations between specific genes and the neural pathways that mediate individual differences in both normal and abnormal human behaviors [Hariri et al. 2006]. Previous studies have demonstrated the critical role of the serotonin neurotransmitter system in the development of emotional circuitry and the onset of mood disorders [Ansorge et al. 2004; Lotrich and Pollock 2004]. Specifically, a polymorphism in the human serotonin transporter (5-HTT) gene (5-HTTLPR or SLC6A4) associated with 5-HTT protein expression and function has been shown to modulate the influence of stressful life events on depression and the responses of the amygdala to negative stimuli [Kendler et al. 2005]. Expression of the serotonin transporter (5-HTT) and serotonin uptake was likely to be reduced in individuals who carry a short (s) allele of the serotonin transporter gene (5-HTTLPR), compared to long-allele homozygotes (l/l). Evidence from several other studies consistently indicates increased activation in the amygdala in response to negative stimuli in healthy individuals who carry the short allele (s) versus healthy individuals carrying the homozygous long alleles (l) [Bertolino et al. 2005]. Thus, higher levels of body dissatisfaction and drive for thinness may be associated with the (s) allele of the serotonin transporter gene (5-HTTLPR).

Given neurological pathways, findings to date have suggested some specific patterns of activation in response to body image stimuli. The fusiform gyrus has suggested to be implicated in neural processes related to face and body recognition.

Patients with body dysmorphic disorder (BDD) have shown increased left hemisphere activation in the prefrontal cortex and temporal lobe when examining faces. It suggests that the BDD patients are more detail oriented in face processing than controls.

Genetic and biological influences are likely to place certain individuals at increased risk for body image disturbances but only in the face of environmental stimuli (cultural, familial, or peer influences). Thus, the genetic-environemental interactions play an important role in the development of body image. For example, two individuals may be exposed to the same risk factor (e.g. appearance media exposure) but only one individual may develop body dissatisfaction in response to this risk. The individual who develops body dissatisfaction may have higher genetic risk that becomes activated in the face of psychosocial stressor.

A further approach through which human behaviour related to appearance, attractiveness, and body image may be understood is that of evolutionary perspective. The evolutionary approach provides an explanation of the psychological mechanisms which have evolved to drive human behaviour toward physical attractiveness [Tooby, Cosmides 2005]. Specifically, under the heading of sexual selection, "…evolutionary psychologists have suggested that some traits in humans

evolved, not because they confer any survival advantage but rather because they are related to fitness-enhancing benefits or because they are used in competition for access to potential mates. In other words some traits are perceived and used by individuals to access the quality and desirability of potential mates in order to enhance their own chances of reproductive success." [Swami 2011:20].

The fitness-related evolutionary theories, including human mate selection, good genes and differential parental solicitude, posit that humans have evolved universal standards of attractiveness that are based on evidence of health and reproductive fitness [Barber 1995; Buss, Schmitt 1993]. A central statement of the mate-selection theory is that attractiveness is distinctive between the male and female [Buss 1998]. According to this theory, in humans, attractiveness and showiness is more important for female. Men seek this particular alluring characteristic of women because they believe it signals youth and reproductive fitness [Buss 1998]. In contrast, women look for men with resources rather than attractiveness, because such men are able to provide for their intended offspring. According to Buss [1999], mate-selection research focuses on preferences rather than behaviour, even though mate-choice is a behavioural outcome of mate-preferences.

The good-genes theory predicts that an attractive appearance should be meaningful in human interactions because attractiveness is a direct visual cue to mate quality, health and heterozygosity [Thornhill, Gangestad 1999; Barber 1995]. Thus, good-genes theory allows predicting differential judgment and treatment as a function of attractiveness because perceivers have evolved to prefer attractive people for their good health. Given good health is important to survival, attractiveness should be equally relevant and important to both sexes when making a choice [Thiessen 1996].

Differential parental solicitude theory (a derivative of Trivers's [1972] parental investment theory) states that to enhance their own reproductive success, parents invest differently in their offspring depending on each child's fitness, quality and reproductive potential [Buss 1999]. Parents and other adults should allocate more effort, resources, and care into the well being of offspring. If attractiveness is an indicator of quality, parents should invest more in attractive than unattractive offspring. Thus, more attractive offspring will presumably be treated more favourably than an unattractive offspring [Buss 1999, Langlois et al. 1995].

In addition, differences in health and quality should be manifested by differences in behaviour in such a way that attractive individuals should exhibit more positive behaviour than their unattractive counterparts. Given this theory equally validates the health and quality in boys and girls, gender differences are not indicated in how boys and girls are judged and treated as a function of attractiveness [Daly, Wilson 1995].

## Body image in childhood and adolescence

Recent studies have suggested that boys and especially girls, become critical of their bodies before adolescence. Williamson and Delin [2001] have presented evi-

dence that 5-year old girls show a significant tendency to prefer thinner figures than their current size. Those girls were likely to be less satisfied with their body size than 5-year old boys.

There is growing evidence that by the age 6, and probably earlier, children are aware of the societal bias against fat people and will frequently express this bias themselves. Overweight children begin to internalize this message and wish they were thinner. This aversion to fat people may increase with age [Cash, Smolak 2011].

Adolescence is an important period for the development of body image. The transition to adulthood that begins with pubertal changes in physical traits, emerging sexuality, and beginning of psychosocial maturity, is more stressful for girls than boys. Unlike boys, girls tend to confront more of these demands simultaneously or in rapid sequence. Additionally, girls as a group experience more limited options for success in various realms [Cash, Pruzinsky 2004]. These negative experiences are associated with poor body image and higher levels of drive for thinness and dieting. Although little is known about the development of body image in adolescent boy, it seems that the timing of puberty does not have a strong or lasting effect on boys' body image.

In Western cultures, the perception of overall physical appearance is one of the most important components of an adolescent's global self-esteem. Low self-esteem and negative affect are correlated with negative body image, and this correlation is significantly greater for girls than boys. The tendency of adolescent girls to feel less positively about their changing body's shape is a significant risk factor for eating disorders and depression. The well-documented tendency for girls to be more depressed than boys, begin around age 14 [Cash, Pruzinsky 2004].

## Aim of the study

Given the importance of body image for the quality of life, a study was designed to address this phenomenon. The main objective was to assess body image (dis)satisfaction in adolescent males and females depending on the socioeconomic status (SES) of their families and their own lifestyle.

# Materials and methods

## Subjects

Subjects were derived from the ADOPOLNOR project, a prospective, cross-sectional survey of health and quality of life in adolescents. A total of 3130 subjects, aged 13–18, were studied.

The groups were categorized by gender and comprised 1552 boys and 1578 girls. In addition, they were stratified by family socio-economic status (SES) and their own lifestyle.

## Study design

This survey was carried out between October 2009 and February 2010 in lower and upper secondary schools in Wielkopolska province. Semi-structured, self-reported questionnaires were administered to students and their parents. Students were asked about their lifestyle and perceived overall body appearance (body image). Parents reported on their socio-economic status. Measurements were taken for students' somatic traits of which the stature and body mass were used in the present study.

## Variables

The socio-economic status of families was established on the basis of the subjective assessment of family income and mother's educational attainments. In addition, urbanization factor was included in a set of social characteristics. Each characteristic was categorized as binary or multistate categorical variables.

The subjective assessment of family income included following responses: (1) not sufficient at all, (2) sufficient but only to a very limited degree, (3) sufficient to a degree, (4) sufficient, (5) more than sufficient). They were categorized according to a dichotomous scale: (1) sufficient income and (2) insufficient income. The level of the mothers' education was divided into three groups: (1) university-level, (2) secondary, (3) primary and vocational education. The urbanization factor was categorised into three groups: (1) city > 100,000 inhabitants, (2) city < 100,000 inhabitants, (3) village.

Lifestyle was defined on the basis of health-related behaviour choices as declared by the studied adolescents: smoking – (1) no, never, (2) quit, past smoker, (3) still smoking, current smoker; physical activity (answer to the question: How many times over the last 7 days have you engaged in intense physical activity? (1) 1–4 and more, (2) 1–3, (3) not at all; and sexual activity (answer to the question: Have you had a sexual intercourse with another person? (1) no, never, (2) yes, once, (3) yes, more than one.

For the purposes of the present study, body dissatisfaction was defined after Grogan [2008:19] "…as a person's negative thoughts and feelings about his or her body".

Global dissatisfaction with body size was assessed using figural ratings technique. The figural method is a quantitative measure of degree and direction of body dissatisfaction [Grogan 2008:26; Thompson et al. 1999:52–57].

Validation of the figural ratings scale, using the test-retest reliability study, has shown quite high reliability scores, ranging from 0.71 to 0.92 [Thompson, Altabe 1991].

This method involves using a broad range of figures ranging in size from very thin to very overweight. These figures can be schematic outlines or silhouettes of the human form.

The 9 female and 9 male silhouette drawings used in the study were developed by Stunkard et al. [1983] to determine the weight status of adoptive children, and then subsequently used by Fallon and Rozin [1985] to consider body perception (Fig. 1).

Participants were asked to select one from nine figures varying in size from underweight/very thin (1) to very heavy/overweight (9) and answer two questions: "Which of the following figures best reflects your current appearance?" and "Which of the following figures is the figure would you like to have?". Then the subjects marked the number corresponding to the figure which they believed best reflected their current body size as well as the figure which they regarded as ideal. The body dissatisfaction was calculated from the figure ratings as the discrepancy between the two ratings, the current and ideal figures.

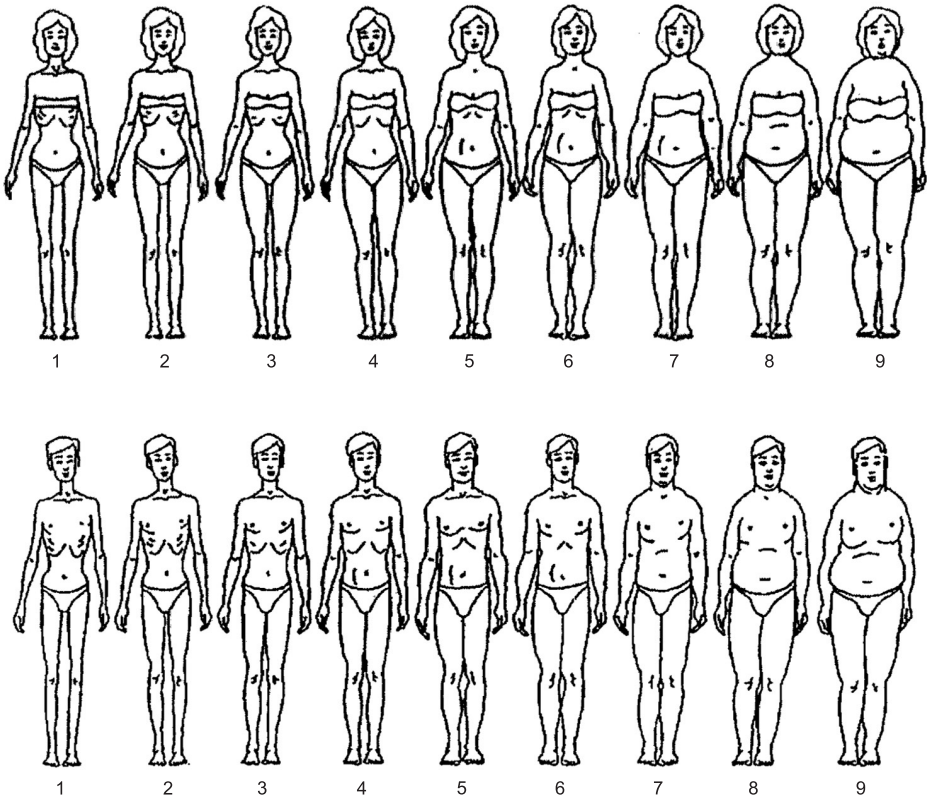



Fig. 1. The female and male silhouette templates used in the figural ratings technique. See text for explanation

Body image was controlled for BMI (the body weight in kilograms divided by the square of the height in meters kg/m$^2$). The BMI criterion was categorised in accordance with Cole's cut-off points into underweight, normal, overweight and obese weight status [Cole et al. 2000, 2007].

## Data analysis

In order to establish the level of the subjects' satisfaction or dissatisfaction with their own bodies, the difference between the subjective own body assessment and the body chosen from among the presented silhouettes as ideal was calculated. The difference, given in absolute values, defined the level of satisfaction (0) or dissatisfaction (1–8) with the subjects' own bodies [Swami et al. 2010]. The data were then analysed by means of descriptive statistics and the logistic regression models.

All statistical computations were performed using STATISTICA data analysis software system, version 9.0 (2009) www.statsoft.com. The level of significance was set at $p=0.05$.

# Results

## Group description

Table 1 presents information for the demographic, social and lifestyle behaviour characteristics for the sample.

For the vast majority of parents (84.5%), the income of their families was sufficient to satisfy living needs. An analysis of data concerning physical activity revealed that most adolescents considered themselves to be physically active. Slightly over 46% of respondents reported their engagement in intense physical activity, as many as 4 or more times a week, similar number (47.6%) of respondents exercised for 1–3 times a week, whereas only 5.5% did not take part in any intense physical activity at all. Boys turned out to be more physically active than girls ($p<0.001$).

A vast majority of the respondents did not smoke (79.9%). Almost seven percent (6.6%) of respondents had quit smoking before the survey, and 13.6% of boys and girls said they were still

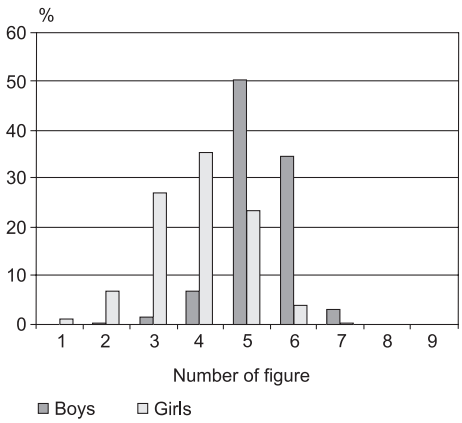



Fig. 2. Percentage distribution of the body ideal in the adolescent females and males

Table 1. Sample characteristics

| Variable | Boys (N=1552) | | Girls (N=1578) | |
|---|---|---|---|---|
| | n | % | n | % |
| Age (in years) | | | | |
| 13 | 107 | 6.9 | 140 | 8.9 |
| 14 | 272 | 17.5 | 255 | 16.2 |
| 15 | 269 | 17.3 | 307 | 19.4 |
| 16 | 275 | 17.7 | 331 | 21.0 |
| 17 | 287 | 18.5 | 231 | 14.6 |
| 18 | 342 | 22.0 | 314 | 19.9 |
| BMI | | | | |
| Underweight | 126 | 8.1 | 190 | 12.0 |
| Normal | 1127 | 72.6 | 1177 | 74.6 |
| Overweight/Obesity | 270 | 17.4 | 191 | 12.1 |
| Place of residence | | | | |
| City > 100,000 population | 346 | 22.3 | 387 | 24.5 |
| City < 100,000 population | 459 | 29.6 | 478 | 30.1 |
| Village | 679 | 43.7 | 641 | 40.6 |
| Mother's education | | | | |
| Academic | 282 | 18.2 | 289 | 18.3 |
| Secondary | 598 | 38.5 | 579 | 36.7 |
| Primary/Vocational | 589 | 37.9 | 653 | 41.4 |
| Income | | | | |
| Sufficient | 1328 | 85.6 | 1318 | 83.5 |
| Nonsufficient | 163 | 10.5 | 219 | 13.9 |
| Physical activity | | | | |
| 4 and more times/week | 878 | 56.6 | 587 | 37.2 |
| 1–3 times/week | 635 | 40.9 | 854 | 54.1 |
| None | 51 | 3.3 | 121 | 7.7 |
| Smoking cigarettes | | | | |
| No, never | 1240 | 79.9 | 1260 | 79.8 |
| Past smoker | 108 | 7.0 | 99 | 6.3 |
| Current smoker | 212 | 13.7 | 214 | 13.6 |
| Sexual activity | | | | |
| No, never | 1267 | 81.6 | 1363 | 86.4 |
| Yes, one | 75 | 4.8 | 49 | 3.1 |
| Yes, more than one | 193 | 12.4 | 138 | 8.7 |

smoking. There were no significant differences in the prevalence of smokers among girls and boys.

The studied adolescents came from large cities, with over 100,000 inhabitants (23.4%), 29.9% of them came from medium-to-small sized cities (less than 100,000 inhabitants), and 42% lived in villages.

With regard to mother education level, the largest group (39.7%) had either primary or vocational education, the next were mothers with secondary-level education (37.6%), and 8.2% of the inquired mothers said they had university-level education.

As to the body size, expressed in terms of the BMI criterion, it appeared that 72.6% of boys and 74.6% of girls were within the BMI normal. Twelve percent of girls and 8.1% of boys were underweight. Boys (17.4%) were more often overweight or obese than girls (12.1%). There were differences between the two genders with regard to the desired body size e.g. body ideal. Results of figural ratings revealed that boys, more than girls, mostly tended to choose larger bodies (number of silhouettes ranges from 4 onwards) as ideal (37.6% vs. 8.3%, $p<0.001$). Unlike boys, girls were more likely to chose thin bodies (number of silhouettes between 1 and 3) as their ideals (70.5% vs. 5%, $p<0.001$). Histograms shown in Figure 2 corroborate well with this finding.

Girls, significantly more often than boys, believed their bodies to be too large (39.6% vs. 14.3%, $p<0.001$). On the other hand, boys believed, significantly more often than girls, that their bodies were too small (21.6% vs. 8.5%, $p<0.001$).

Having analysed the average level of satisfaction/dissatisfaction with body size in terms of age for boys and girls, it schould be pointed to a significant difference between the genders. In all age groups, the level of dissatisfaction with body size was significantly higher ($p<0.001$) among girls than among boys (Fig. 3).

An analysis of the level of satisfaction with body size depending on the BMI category showed that overweight and obese subjects were the most dissatisfied

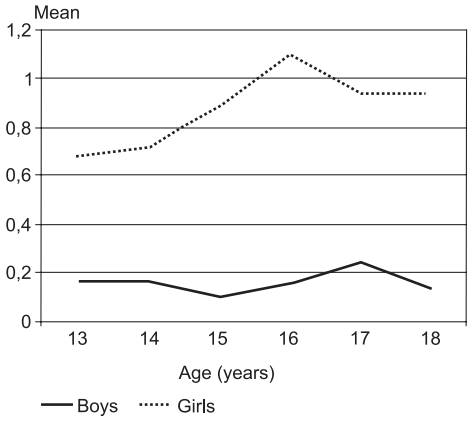



Fig. 3. Average levels of the body image dissatisfaction by gender and age

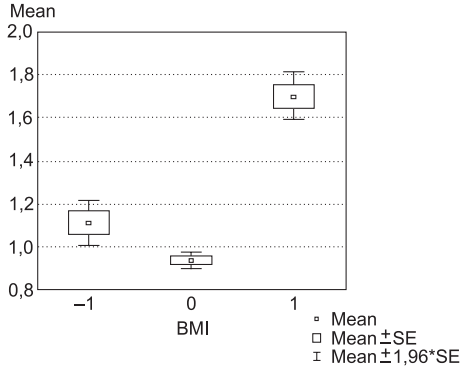



Fig. 4. Mean scores of the body image dissatisfaction in a combined sample of females and males by the BMI criterion: (–1) underweight, (0) normal, (1) overweight/obese

with body size (average satisfaction level score was 1.7) and were followed in this respect by underweight subjects with mean score1.1. The least dissatisfied were subjects within the BMI norm al with mean score 0.9. These differences were statistically significant ($F=118.7$, $p<0.001$) (Fig. 4).

## Social and cultural correlates of the body image satisfaction

In order to assess the impact and establish the structure of biological variables and variables related to lifestyle and socio-economic conditions on the satisfaction or dissatisfaction with body size, the researchers used a logistic regression analysis. The category of own body perception was transformed to make it possible to obtain a dichotomous distribution of the variable: (0) satisfied, (1) dissatisfied. The aim of the study was to establish whether and to what extent socio-economic status, and lifestyle affected the level of satisfaction with own body among adolescents. A bivariate logistic regression analysis revealed a significant influence of BMI, gender ($p<0.001$) as well as lifestyle variables (physical activity, smoking, sexual activity: $p<0.05$) on the level of satisfaction with body size (Table 2), with the BMI proving to be the strongest in affecting the level of own body satisfaction. Variables found in bivariate logistic regression models to be significantly associated with body image satisfaction were included in the multivariate logistic regression models. The variables related to the subjects' socio-economic status turned out not to have any significant impact on the way young people perceived their bodies.

In addition, the OR, i.e. the odds on dissatisfaction with body size occurring in adolescents were calculated. The OR value characterises the likelihood of an event in various groups. Table 2 contains the OR values for various potential predictors

Table 2. Predictors of body size satisfaction/dissatisfaction – results of logistic regression

| Variable | All – predictor model | | | | Adjusted model | | | |
|---|---|---|---|---|---|---|---|---|
| | Estimated parameter value | OR | 95% Cl | *p* | Estimated parameter value | OR | 95% Cl | *p* |
| Gender | 0.505 | 1.66 | 1.40–1.96 | <0.001 | 0.512 | 1.67 | 1.42–1.96 | <0.001 |
| BMI | 0.446 | 1.56 | 1.32–1.85 | <0.001 | 0.447 | 1.56 | 1.33–1.84 | <0.001 |
| Physical activity | 0.258 | 1.29 | 1.12–1.50 | <0.001 | 0.216 | 1.24 | 1.08–1.42 | 0.002 |
| Smoking | 0.135 | 1.14 | 1.00–1.31 | 0.048 | 0.137 | 1.15 | 1.01–1.30 | 0.033 |
| Income | 0.129 | 1.14 | 0.87–1.49 | 0.349 | | | | |
| Mother's education | –0.016 | 0.98 | 0.87–1.11 | 0.790 | | | | |
| Sexual activity | –0.138 | 0.87 | 0.76–1.00 | 0.055 | –0.172 | 0.84 | 0.73–0.96 | 0.011 |
| Place of residence | –0.056 | 0.84 | 0.85–1.05 | 0.310 | | | | |

OR – odds ratio
Cl –95% +95%: lower and upper limit of 95-percent confidence interval for odds ratio

of dissatisfaction with body size. It shows that the risk of dissatisfaction with body size in girls was almost twice as high as in boys (OR=1.67). In addition, a higher BMI value meant a higher risk of dissatisfaction with the body (OR=1.56).

The subjects characterised by lower physical activity were at greater risk of dissatisfaction with body size in comparison with the subjects that were more physically active (OR=1.24). Smoking was another factor lowering the satisfaction with one's own body (OR=1.15). Moreover, smoking was a stronger determinant of dissatisfaction with body size in girls than in boys. The odds quotient of body dissatisfaction in girls ranged from 1.47 for those who admitted to smoking in the past to 2.11 for current smokers. It was also established that the type of sexual activity affected the level of satisfaction with body size. Sexually active subjects had a positive image of their own bodies (OR=0.84).

Having taken into account all the predictors in the logistic regression analysis, it was concluded that the factors independently predisposing the subjects to negative body size perception were: female gender, overweight/obesity, lack of physical activity and smoking. Sexual activity has suggested to be associated with a positive perception of body size. The socio-economic status variables do not have any significant impact on the way young people perceive their bodies.

## Discussion

The aim of the study was to establish whether and to what extent lifestyle, socio-economic status and biological features affected the level of satisfaction with own body among adolescents.

The analyses carried out by the authors showed significant differences between boys and girls in own body perception. This result supporty our previous findings and is confirmed by the available literature on the subject [Hargreaves and Tiggemann 2004, Storvoll et al. 2005, Kaczmarek, Durda 2008]. In these studies, just like in the present study, girls were more likely to imagine their own bodies as larger than they were in reality. Responding to cultural norms of male attractiveness, boys expressed their conviction that their bodies were slimmer than they were in reality. This result seems to be a direct consequence of the influence of the media promoting the ideal of a slim woman and large, muscular man as well as the relationships with parents and peers [Lokken et al. 2003]. The discrepancy between the low subjective assessment of one's own body and the unattainable ideal arouses strong negative feelings. These, in turn, may prompt unhealthy behaviour – smoking or abandoning physical activity. In the present study smoking as well as a lack of physical activity displayed a positive correlation with a negative perception of one's own body. The subjects who admitted to smoking had a more negative perception of their bodies. This result is confirmed by studies of other authors [King et al. 2000, Clark et al. 2005].

The present study also demonstrated that girls who admitted to smoking were more dissatisfied with their body size than boys who smoked; this, too, conforms

to findings recorded in the available literature [Boles and Johnson 2001, Potter 2004]. The link between dissatisfaction with one's own body and smoking can be explained by the fact that people who do not feel confident about their own bodies may try to boost their self-confidence by opting for behaviour which they believe will make them more attractive to other members of their group. It should be noted that at the beginning of puberty the role of peers in the functioning of individuals and in their perception of themselves becomes disproportionately bigger than it was in childhood; that is why the concern about one's bodily imperfections in the eyes of others seems to be particularly strong. In addition, there is a belief that smoking affects weight loss. Thus, people who regard their bodies as too large in comparison with the ideal they would like to achieve may start smoking in order to make it easier for themselves to achieve that bodily ideal [Clark et al. 2005]. Given the fact that girls are more dissatisfied with their bodies, they may be more eager to try to find ways to deal with the problem.

The present study also demonstrated the impact of physical activity on the perception of body size. These conclusions are confirmed by studies of many other authors [Anton et al. 2000]. Physical training affects body size, which is why people who often engage in physical activity may be more satisfied with the size of their bodies than people who give up this activity. The results may also suggest that adolescents who do not accept their bodies may avoid physical activity because they are afraid of being judged by others [Bezner et al. 1997]. We can also suspect that the current ideals of attractiveness are so unrealistic and so hard to attain that this leads to considerable frustration and discourages adolescents from taking up any activity whatsoever. The available literature on the subject also contains results contrary to those obtained in the present study, i.e. results suggesting a lack of any link between the level of satisfaction with the body and the level of physical activity [Dunkan et al. 2004].

The present study has demonstrated a link between the level of satisfaction with body size and sexual activity. Those respondents that had a positive image of their own bodies were more likely to have undergone sexual initiation. Similar results were obtained by Cash and Fleming in their research [2002]. They explained this link by the fact that a positive image of one's body resulted in a belief that it was perceived just as positively by others, which in turn boosted self-confidence in interpersonal contacts, thus increasing the likelihood of forming romantic relationships. Yet opinion is divided on this issue. Valle and colleagues [2009] demonstrated that among the adolescents analysed by them a low level of satisfaction with body size predestined the subjects to an early start of sexual activity. However, the link between the level of satisfaction with one's own body and sexual activity of adolescents is not yet well established and requires further research.

In the present study, none of the variables related to the socio-economic status proved to have a significant impact on the level of satisfaction with body size. The lack of a link between the SES variables and satisfaction with one's own body is confirmed by studies conducted by Story and associates [1995]. However, the available literature on the subject contains results that can be regarded as completely opposite. In other studies carried out by O'Dea and Caputi [2001] a low

socio-economic status was correlated with a worse perception of one's own body in adolescents. On the other hand, Paxton et al. [1994] obtained very different results. According to them, a low socio-economic status was correlated with a higher level of satisfaction with one's own body, because the pressure on adolescents with lower SES to develop an ideal body was not as strong.

## Conclusion

On the bases of the results obtained in this study, the follwing conclusion can be drawn.

An important role in the development of body size image in children and adolescents is played by the lifestyle variables (smoking, physical activity and sexual activity). The factors that are the strongest determinants of the perception of one's body are gender and BMI.

## References

Altabe M., Thompson J. K.: Body image changes during early adulthood. *Int J Eat Disorder* 1993; 13(3):323–328.

Ansorge M.S., Zhou M., Lira A., Hen R., Gingrich J.A. (2004): Early-life blockade of the 5-HT transporter alters emotional behavior in adult mice. *Science* 2004; 306:879–881.

Anton S.D., Perri M.G., Riley J.R.: Discrepancy between actual and ideal body images. Impact on eating and exercise behaviors. *Eat Behav* 2000; 1:153–160.

Barber N.: The evolutionary psychology of physical attractiveness: Sexual selection and human morphology. *Ethol Sociobiol* 1995; 16:395–424.

Bertolino A., Arciero G., Rubino V., Latorre V., De Candia M., Mazzola V., et al.: Variation of human amygdala response during threatening stimuli as a function of 5'HTTLPR genotype and personality style. *Biol Psychiatry* 2005;57:1517–1525.

Bezner J.R., Adams T.B., Steinhardt M.A.: Relationship of body dissatisfaction to physical health and wellness. *Am J Health Behav* 1997; 21:147–155.

Boles S., Johnson P.: Gender, weight concerns, and adolescent smoking. *J Addict Dis* 2001; 20(2):5–14.

Brouwers M.: Treatment of body image among women with bulimia nervosa. *J Couns Dev* 1990; 69(2):144–147.

Buss D.M.: Sexual Strategies Theory: Historical origins and current status. *J Sex Res* 1998;34:19–31.

Buss D.M., Schmitt D.P.: Sexual strategies theory: An evolutionary perspective on human mating. *Psychol Rev* 1993; 10:204–232.

Cash T.F., Pruzinsky T.: (Eds.) *Body Images: Development, Deviance and Change*. 1990. New York: Guilford Press.

Cash T.F., Pruzinsky T.: (Eds.) *Body Image A Handbook of Theory, Research and Clinical Practice*. 2004. New York London: The Guilford Press.

Cash T.F., Smolak L.: Understanding body images: historical and contemporary perspectives. In: T.F. Cash and L. Smolak (Eds.) *Body Image A Handbook of Science, Practice, and Prevention* 2nd ed. 2011; 3–12. New York: Guilford Press.

Clark M.M., Croghan I.T., Reading S., Schroeder D.R., Stoner S.M., Patten C.A., Vickers K.S.: The relationship of body image dissatisfaction to cigarette smoking in college students. *Body Image* 2005; 2:263–270.

Cole T.J., Bellizzi M.C., Flegal K.M., Dietz W.H.: Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 2000; 320:1240–1243.

Cole T.J., Flegal K.M., Nicholls D., Jackson A.A.: Body mass index cut offs to define thinness in children and adolescents: international survey. *BMJ* 2007; 335:194–201.

Daly M., Wilson M.: Discriminative parental solicitude and the relevance of evolutionary models to the analysis of motivational systems. In: M.S. Gassaniga (Ed.) *The Cognitive Neurosciences* 1995; 1269–1286. Cambridge, MA: MIT Press.

Dunkan M.J., Al-Nakeeb Y., Nevill A., Jonec M.V.: Body image and physical activity in British secondary school children. *Eur Phys Educ Rev* 2004; 10(3):243–260.

Fallon A.E., Rozin P.: Sex differences in perceptions of desirable body shape. *J Abnor Psychol* 1985; 94:102–105.

Field A.E., Camargo C.A., Taylor C.B., Berkey C.S., Colditz G.A.: Relation of peer and media influences in the development of purging behaviours among preadolescent and adolescent girls. *Arch Paediatr Adolesc Med* 1999; 153:1184–1189.

Fisher S.: The evolution of psychological concepts about the body. In: T.F. Cash and T. Pruzinsky (Eds.) *Body Images: Development, Deviance and Change*. 1990; 3–20. New York: Guilford Press.

Grogan S.: *Body Image: Understanding Body Dissatisfaction in Men, Women, and Children* 2nd ed. 2008. London and New York: Routledge Taylor and Francis Group.

Hariri A.R., Drabant E.M., Weinberger D.R.: Imaging genetics: Perspectives from studies of genetically driven variation in serotonin. *Biol Psychiatry* 2006; 59:888–897.

Hargreaves D.A., Tiggemann M.: Idealized media images and adolescent body image: "comparing" boys and girls. *Body Image* 2004; 1:351–361.

Jackson L.A.: Physical attractiveness. A sociocultural perspective. In: T.F. Cash and T. Pruzinsky (Eds.) *Body Image A Handbook of Theory, Research and Clinical Practice*. 2004; 13–22. New York: Guilford Press.

Jones D.C., Vigfusdottir T.H., Lee Y.: Body image and the appearance culture among adolescent girls and boys: An examination of friend conversations, peer criticism, appearance magazines, and the internalisation of appearance ideals. *J Adolesc Res* 2004; 19:323–339.

Kaczmarek M., Durda M., Krzyżaniak A.: Is gender a crucial cause of body image in adolescence? *Pol J Environ Stud* 2008; 17(4A):182–186.

Kendler K.S., Kuhn J.W., Vittum J., Prescott C.A., Riley B.: The interaction of stressful life events and a serotonin transporter polymorphism in the prediction of episodes of major depression: A replication. *Arch Gen Psychiatry* 2005; 62:529–535.

King T., Matacin M., White K., Marcus B.: A prospective examination of body image and smoking cessation in women *Body Image* 2005; 2:19–28.

Klump K.L., Suisman J.L., Burt S.A., McGue M., Iacono W.G.: Genetic and environmental influences on disordered eating: An adoption study. *J Abnorm Psychol* 2009; 118: 797–805.

Langlois J. H., Ritter J.M., Casey R.C., Sawin D.B.: Infant attractiveness predicts maternal behavior and attitudes. *Dev Psychol* 1995; 31:462–472.

Lokken, K., Ferraro, F.R., Kirchner, T., Bowling M.: Gender differences in body size dissatisfaction among individuals with low, medium, or high levels of body focus. *J Gen Psychol* 2003; 130:305–310.

Lotrich F.E., Pollock B.G.: Meta-analysis of serotonin transporter polymorphisms and affective disorders. *Psychiatr Genet* 2004; 14:121–129.

O'Dea J.A., Caputi P.: Association between socioeconomic status, weight, age and gender, and the body image and weight control practices of 6- to 19-year-old children and adolescents. *Health Educ Res* 2001; 16(5):521–532.

Paxton S.J., Sculthorpe A., Gibbons K.: Weight-loss strategies and beliefs in high and low socioeconomic areas of Melbourne. *Aust J Public Health* 1994; 18:412–417.

Potter B.K., Pederson L.L., Chan S.S., Aubut J.A., Koval J.J.: Does a relationship exist between body weight, concerns about weight, and smoking among adolescents? An integration of the literature with an emphasis on gender. *Nicotine Tob Res* 2004; 6(3):397–425.

Pruzinsky T., Cash T.F.: Understanding body images. Historical and contemporary perspectives. In: T.F. Cash and T. Pruzinsky (Eds.) *Body Image. A Handbook of Theory, Research, and Clinical Practice*. 2004:3–12. New York, London: The Guilford Press.

Schilder P.: *The Image and Appearance of the Human Body*.- 1935/1950 reprinted in 1999 by Routlege. 1999. The International Library Psychology: 204 Volumes Founded by C.K. Ogden

Schontz F.C.: *Perceptual and Cognitive Aspects of Body Experience*. 1969. New York: Macmillan.

Storvoll E. E., Strandbu Å., Wichstrøm L.: A cross-sectional study of changes in Norwegian adolescents' body image from 1992 to 2002. *Body Image* 2005; 2:5–18.

Story M., French S.A., Resnik M.D., Blum R.W.: Ethnic/racial and socioeconomic differences in dieting behaviors and body image perceptions in adolescents. *Int J Eat Disord* 1995; 18:173–179.

Swami V.: Evolutionary perspectives on human appearance and body image. In: T.F. Cash and L. Smolak (Eds.) *Body Image A Handbook of Science, Practice, and Prevention* 2nd ed. 2011:20–29. New York: Guilford Press.

Swami V., Begum S., Petrides K.V.: Associations between trait emotional intelligence, actual – ideal weight discrepancy, and positive body image, *Pers Indiv Differ* 2010; 49: 485–489.

Thiessen D.: *Bittersweet Destiny: The Stormy Evolution of Human Behavior*. 1996. New Brunswick, NJ: Transaction 1996.

Thompson J. K., Altabe M. N.: Psychometric qualities of the Figure Rating Scale. *Int J Eat Disorder* 1991; 10:615–619.

Thompson J.K., Heinberg L.J., Altabe M., Tantleff-Dunn S.: . 1999. Washington, DC: American Psychological Association.

Thornhill R., Gangestad S.W.: The scent of symmetry: a human sex pheromone that signals fitness? *Evol Hum Behav* 1999; 20:175–201.

Tiggemann M.: Social perspectives on human appearance and body image. In: T.F. Cash and L. Smolak (Eds.) *Body Image A Handbook of Science, Practice, and Prevention* 2nd ed. 2011:12–20. New York: Guilford Press.

Tooby J., Cosmides L.: Conceptual foundations of evolutionary psychology. In: D.M. Buss (Ed.) *The Handbook of Evolutionary Psychology* 2005:5–67. Hoboken, NJ: Wiley.

Wade T.D., Bulik C.M., Heath A.C., Martin N.G., Eaves L.J.: The influence of genetic and environmental factors in estimations of current body size, desired body size, and body dissatisfaction. *Twin Research and Human Genetics* 2001; 4:260–265.

Williamson S., Delin C.: Young children's figural selections: Accuracy of reporting and body size dissatisfaction. *Int J Eat Disorder* 2001; 29(1):80–84.

# Health Problems and Chronic Conditions

Alicja Krzyżaniak, Maria Kaczmarek,
Barbara Stawińska-Witoszyńska,
Małgorzata Krzywińska-Wiewiorowska,
Magdalena Skrzypczak

# Chronic diseaes and disabling conditions in children and addescents from Wielkopolska province according to secondary sources of information

**Abstract:** Health status of children and adolescents in the Wielkopolska province has been analysed based on secondary sources of information. It has been concluded that over the period analysed in the study, accidents and injuries were the most frequent causes of deaths among adolescents. Allergies, permanent musculoskeletal disorders and deforming dorsopathies were the most frequently recorded health problems in the pre-adult population. Congenital cardiac and musculoskeletal anomalies proved to be of the highest incidence of all congenital malformations. Leukaemia as well as malignant brain and central nervous system neoplasms accounted for the highest proportion of neoplasm cases in children and adolescents. The study also shows that the implementation of the obligatory vaccination programme against certain infectious diseases resulted in a significant decrease in the incidence of these diseases in children and adolescents. Finally, it has been concluded that there is a need for a more standardized method of collecting statistical data concerning the health status in the examined group.

**Key words:** secondary sources of information, children, adolescents, health status

## Introduction

Health status of children and adolescents in a given area is a result of interaction of numerous factors, among which the socio-economic factor and access to quality healthcare are believed to be the most significant ones. The essence of proper healthcare offered in a given population is to be aware of current health status of this population. Child and adolescent healthcare focuses mainly on preventive measures and on detecting diseases and anomalies, especially those that may lead

to serious complications or may develop into serious health problems in children's or adolescents' future lives. Diseases and anomalies should be detected during prevention checkups that is to be performed by a general practitioner after the current healthcare system has been restructured. The focus on prevention in child and adolescent healthcare results from specific biological features of a developing organism and its susceptibility to environmental and genetic factors. Methodological limitations on the evaluation of changes in health status of a given population require the researchers to use indicators that are both common and comparable with one another. As developing accurate and direct measures of health of a population is extremely difficult, measures that constitute indirect indicators of health are employed. The estimation of the incidence of diseases and injuries, and their consequences such as various forms of disability or deaths, serves at the same time to assess the general health status of the population [Jędrychowski 1999]. Data gathered from the so called secondary sources of information, i.e. medical records from in-patient and out-patient medical centres, copies of death certificates issued for statistical purposes, records of the Polish Social Insurance Institution (*ZUS*) together with statistical data obtained from the Polish Central Statistical Office (*GUS*), make it possible to obtain three negative measures of health: incidence as well as morbidity and mortality rates.

In line with the World Health Organisation guidelines, the evaluation of the health status of a specific population should not be based only on up-to-date cross-sectional research, but also on data that had been originally collected for purposes other than epidemiological study. We believe that the presentation of children and adolescent's health status in the Wielkopolska province based on secondary sources of information is a valuable supplement to what is already known about the health of the young population, part of which has been examined under the Polish-Norwegian ADOPOLNOR programme.

The aim of this study was to analyse children's and adolescents' health status in the Wielkopolska province based on secondary sources of information.

## Material and methods

Data used in the analysis has come from numerous sources. Wielkopolska Regional Centre for Public Health has provided the data on major causes of mortality as well as on the incidence of chronic diseases and anomalies observed in children and adolescents. The Voivodeship Sanitary and Epidemiological Station in Poznań together with the Department of Epidemiology of the National Institute of Public Health have provided the data on the incidence of contagious diseases among children. Information on the incidence of congenital malformations has come from the Polish Registry of Congenital Malformations, created by Prof. A. Latos-Bieleńska and Dr. A. Materna-Kiryluk. Data on the incidence of malignant tumour in children and adolescents in the Wielkopolska province and across Poland have been cited from the works of the Institute of Oncology, Warsaw and the

Greater Poland Cancer Centre [Dyzmann–Sroka et al. 2006–2009; Wojciechowska et al. 2006–2009].

Due to a high diversity of sources used for the purpose of this study, with age groups and periods of observation varying from source to source, the data have been presented in a variety of forms. The incidence of various diseases, as well as morbidity and mortality rates, have been presented in accordance with the source materials, in numbers and percentage. The incidence of congenital malformations in children below 2 years old in the Wielkopolska province has been presented by a number of children affected per 1,000 births for every district in the province. The age-specific mortality rate was calculated for children aged 10–19. The proportions of major causes of deaths in cancer patients both nationally and in the Wielkopolska province have been presented for the group aged 0–19. The incidence of child infectious diseases, influenza, hepatitis B as well as C, and salmonelloses in various age groups in the years 2006–2008 has been presented in the forms of maps or diagrams. The number of children with congenital malformations per 10,000 births for particular districts in the Wielkopolska province in the years 2001–2007 has been presented in the form of maps. The same form has been employed to show the SIR model for malignant tumour incidence in the Wielkopolska province in the years 1999–2006 that has been included in this paper by courtesy of Dariusz Godlewski and Paweł Wojtyś from the Centre of Cancer Prevention and Epidemiology, Poznań [Godlewski, Wojtyś 2009].

## Results and discussion

### Health in children and adolescent aged 0–19 [based on the data from the Wielkopolska Regional Centre for Public Health]

#### Deaths in the group aged 10–19 in the years 2006–2008

The overall number of deaths in the Wielkopolska province in the years 2006–2008 ranged between 29,684 and 31,159. In those years, children and adolescents aged 10–19 accounted for 0.45%, 0.44% and 0.51% of all deaths in respective years. Accidents were the most frequent cause of deaths in the group aged 10–19. In the years 2006–2008, there were 271 deaths caused by accidents in boys accounting for 77.1% of the total number. What was disturbing in those years was a notable increase in the rate of mortality caused by external causes. Table 1 shows causes of deaths for males and females in various age groups. Neoplasms were the second most frequent cause of deaths, accounting for 58 deaths. Nervous system diseases and cardiovascular system disorders were the third and the fourth most frequent causes, respectively. The mortality rate due to circulation system disorders de-

Table 1. Causes of death according to age and sex

| Cause | Age | Year | | | | | | | | | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 2006 | | | 2007 | | | 2008 | | | |
| | | ♂ | ♀ | T | ♂ | ♀ | T | ♂ | ♀ | T | |
| Accidents V01-Y98 | 10–14 | 11 | 5 | 16 | 13 | 3 | 16 | 5 | 5 | 10 | 42 |
| | 15–19 | 46 | 17 | 63 | 62 | 11 | 73 | 61 | 32 | 93 | 229 |
| | total | 57 | 22 | 79 | 75 | 14 | 89 | 66 | 37 | 103 | 271 |
| Neoplasms C00-D48 | 10–14 | 5 | 4 | 9 | 5 | 0 | 5 | 4 | 3 | 7 | 21 |
| | 15–19 | 7 | 7 | 14 | 6 | 5 | 11 | 7 | 5 | 12 | 37 |
| | total | 12 | 11 | 23 | 11 | 5 | 16 | 11 | 8 | 19 | 58 |
| Diseases of the nervous system G00-G99 | 10–14 | 0 | 1 | 1 | 1 | 0 | 1 | 5 | 2 | 7 | 9 |
| | 15–19 | 6 | 2 | 8 | 3 | 3 | 6 | 8 | 1 | 9 | 23 |
| | total | 6 | 3 | 9 | 4 | 3 | 7 | 13 | 3 | 16 | 32 |
| Diseases of the circulatory system I00-I99 | 10–14 | 2 | 2 | 4 | 0 | 1 | 1 | 0 | 1 | 1 | 6 |
| | 15–19 | 8 | 3 | 11 | 3 | 3 | 6 | 3 | 0 | 3 | 20 |
| | total | 10 | 5 | 15 | 3 | 4 | 7 | 3 | 1 | 4 | 26 |
| Diseases of the respiratory system J00-J99 | 10–14 | 1 | 1 | 2 | 1 | 0 | 1 | 1 | 0 | 1 | 4 |
| | 15–19 | 1 | 2 | 3 | 3 | 0 | 3 | 0 | 3 | 3 | 9 |
| | total | 2 | 3 | 5 | 4 | 0 | 4 | 1 | 3 | 4 | 13 |
| Congenital anomalies Q00-Q99 | 10–14 | 1 | 0 | 1 | 1 | 2 | 3 | 0 | 0 | 0 | 4 |
| | 15–19 | 2 | 1 | 3 | 1 | 0 | 1 | 2 | 1 | 3 | 7 |
| | total | 3 | 1 | 4 | 2 | 2 | 4 | 2 | 1 | 3 | 11 |
| Endocrine, nutritional and metabolic diseases E00-E90 | 10–14 | 2 | 1 | 3 | 0 | 1 | 1 | 2 | 0 | 3 | 7 |
| | 15–19 | 2 | 0 | 2 | 0 | 0 | 0 | 0 | 1 | 1 | 3 |
| | total | 4 | 1 | 5 | 0 | 1 | 1 | 2 | 1 | 3 | 9 |
| Certain infectious and parasitic diseases A00-B99 | 10–14 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | 15–19 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 2 | 3 | 4 |
| | total | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 2 | 3 | 4 |
| Diseases of the digestive system K00-K93 | 10–14 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 1 |
| | 15–19 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 1 | 2 |
| | total | 1 | 0 | 1 | 1 | 0 | 1 | 1 | 0 | 1 | 3 |
| Diseases of the genitourinary system N00-N99 | 10–14 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 1 |
| | 15–19 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 |
| | total | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 1 | 2 |
| Symptoms not elsewhere classified R00-R99 | 10–14 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| | 15–19 | 0 | 0 | 0 | 1 | 1 | 2 | 0 | 0 | 0 | 2 |
| | total | 1 | 0 | 1 | 1 | 1 | 2 | 0 | 0 | 0 | 3 |

creased every year (15, 7 and 4 deaths). There were 11 cases of deaths due to congenital malformations. There were also some isolated cases of deaths caused by contagious diseases (4 cases), and diseases of digestive or genitourinary systems.

## The incidence of chronic diseases among children and adolescents in the Wielkopolska province in the years 2006–2008

The incidence of chronic diseases among children and adolescents are presented in Tables 2, 3, 4, 5, 6, and 7.

The diagnosis of the diseases and defects has been presented in line with the 10th Revision of (ICD–10). The incidence of diabetes and thyroid disease is shown in Table 2. In 2008, the number of new cases of mental retardation and epilepsy was lower in comparison with the preceding years, whereas the incidence of cerebral palsy stayed the same over the period discussed in the study (see Table 3). The incidence of diabetes, thyroid diseases, asthma as well as food and skin allergies decreased in comparison with the preceding year (see Tables 2 and 4). The incidence of hypertension among children and adolescents in the years 2007–2008 remained the same (see Table 4). Over the course of the three years under study, the incidence of deforming dorsopathies considerably decreased. In the same period incidence of cancer increased (see Tables 5 and 7). In the years 2006–2008 there was a significant decrease in the number of sight defects diagnoses (see Table 6).

Table 2. Diabetes and disorders of thyroid gland

| Diagnosis according to ICD- 10 | 2006 | 2007 | 2008 |
|---|---|---|---|
| Total | 20353 | 19764 | 15697 |
| Disorders of thyroid gland E00-E07 | 490 | 1132 | 431 |
| Diabetes E10-E14 | 162 | 206 | 185 |

Table 3. Diseases of the central nervous system

| Diagnosis according to ICD-10 | 2006 | 2007 | 2008 | Total |
|---|---|---|---|---|
| Mental retardation F70-F79 | 293 | 310 | 287 | 890 |
| Epilepsy G40 | 381 | 397 | 349 | 1.127 |
| Cerebral palsy G80 | 149 | 148 | 147 | 444 |
| Total | 823 | 855 | 783 | 2.461 |

Table 4. Hypertensive disease, diseases of the genitourinary system, allergies

| Diagnosis according to ICD-10 | | 2006 | 2007 | 2008 | Total |
|---|---|---|---|---|---|
| Hypertensive disease I10-I15 | | 309 | 356 | 356 | 1.021 |
| Diseases of the genitourinary system N00-N23 | | 993 | 987 | 896 | 2.876 |
| allergies | asthma J45 | 2.186 | 2.234 | 2.145 | 6.565 |
| | food hypersensitivity K52.2 | 1650 | 1.699 | 1.633 | 3.492 |
| | dermatitis L27.2 | 1.610 | 1.826 | 1.519 | 4.955 |
| Total | | 5.258 | 7.102 | 6.549 | 18.909 |

Table 5. Permanent musculoskeletal disorders and deforming dorsopathies

| Diagnosis according to ICD-10 | 2006 | 2007 | 2008 | Total |
|---|---|---|---|---|
| Permanent musculoskeletal disorders | 196 | 193 | 183 | 572 |
| Deforming dorsopathies M40-M41 | 3.462 | 2.864 | 2.628 | 8.954 |
| Total | 3.658 | 3.057 | 2.811 | 9.526 |

Table 6. Sight defects and other disorders

| Diagnosis according to ICD-10 | 2006 | 2007 | 2008 | Total |
|---|---|---|---|---|
| Disorders of refraction and accommodation H52 | 2.383 | 2.104 | 1.943 | 6.430 |
| Other patients in need of specialist care | 1.846 | 1.597 | 1.576 | 5.019 |
| Total | 4.229 | 3.701 | 3.519 | 11.449 |

Table 7. Neoplasms and anaemia

| Diagnosis according to ICD-10 | 2006 | 2007 | 2008 | Total |
|---|---|---|---|---|
| Neoplasms C00-C97, D00-D48 | 120 | 113 | 139 | 372 |
| Anaemia D50-D59 | 1.312 | 1.343 | 1.197 | 3.852 |
| Total | 1.432 | 1.456 | 1.336 | 4.224 |

## The incidence of congenital malformations based on the Polish Registry of Congenital Malformations

The incidence of congenital malformations is a major concern mainly for neonatologists and physicians treating the youngest patients. Developments in the field of neonatal care and early detection of such malformations accompanied by new possibilities of treating some of them, lead either to full recovery or to the extension of life expectancy of children with congenital malformations. Although such malformations are not a common health problem for the group aged 10–18, their high incidence in the infancy together with constantly improving methods of treatment suggest that the proportion of children who will undergo treatment is likely to increase. The Polish Registry of Congenital Malformations has been kept since 1997 and it currently covers all Polish provinces. The malformations registered are those found among children up to 2 years of age. The aim of the registry is not only to analyse the incidence of congenital malformations, but also to monitor their incidence and to see in what way early prenatal diagnosis may influence treatment of such conditions. Early diagnosis and prevention by means of folic acid are of high importance especially in cases of neural tube defects.

The incidence of congenital malformations in the years 200–2004 in provinces that are covered by the registry is shown in Table 8.

The highest incidence of congenital malformations per 10,000 births was in the Wielkopolska province. The incidence of particular malformations in the Wielkopolska province in the years 1998–2006 is shown in Table 9.

Table 8. Prevalence (P) of congenital anomalies in voivodships included in Polish Registry of Congenital Malformations in 2000–2004 (on 10 000 births)

| Voivodship | 2000 | | 2001 | | 2002 | | 2003 | | 2004 | | 2005 | | 2006 | | 2007 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | N | P | N | P | N | P | N | P | N | P | N | P | N | P | N | P |
| Dolnośląskie | 379 | 144.4 | x | x | x | x | 392 | 162.6 | 459 | 183.3 | | | | | | |
| Kujawsko- Pomorskie | 337 | 155.8 | 345 | 163.3 | 392 | 193.7 | 359 | 177.5 | 345 | 1711 | | | | | | |
| Lubelskie | x | X | x | X | 476 | 227.7 | 527 | 246.9 | 490 | 234.5 | | | | | | |
| Lubuskie | 166 | 164.7 | 194 | 193.1 | 384 | 194.0 | 178 | 190.8 | 201 | 203.0 | | | | | | |
| Łódzkie | x | X | x | X | x | X | 309 | 141.2 | 342 | 151.4 | | | | | | |
| Mazowieckie | x | X | x | X | x | X | x | X | 870 | 179.1 | | | UNDER REVIEW | | | |
| Opolskie | 252 | 267.5 | 180 | 200.5 | 130 | 152.0 | 140 | 171.0 | 116 | 140.6 | | | | | | |
| Podkarpackie | x | X | x | X | 550 | 256.8 | 511 | 248.1 | 513 | 248.7 | | | | | | |
| Pomorskie | 369 | 156.6 | 362 | 153.9 | 408 | 182.1 | 401 | 178.1 | 385 | 167.7 | | | | | | |
| Śląskie | x | X | 869 | 212.0 | 822 | 207.8 | 742 | 188.5 | 701 | 173.7 | | | | | | |
| Warmińsko- Mazurskie | 403 | 256.9 | 351 | 227.5 | 314 | 210.8 | 225 | 154.4 | 171 | 117.2 | | | | | | |
| Wielkopolskie | 1060 | 300.2 | 943 | 270.9 | 926 | 278.5 | 1132 | 336.7 | 1144 | 339.1 | 985 | 277.9 | 1050 | 286.81 | 657 | |
| Zachodniopomorskie | 309 | 182.0 | 289 | 475.5 | 268 | 169.7 | 213 | 135.0 | 246 | 153.7 | | | | | | |

Table 9. Prevalence of various groups of congenital anomalies in Wielkopolska province (on 10 000 births)

| Type of malformation | 1998 | | 1999 | | 2000 | | 2001 | | 2002 | | 2003 | | 2004 | | 2005 | | 2006 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | N | P | N | P | N | P | N | P | N | P | N | P | N | P | N | P | N | P |
| Q00-07 Congenital malformations of the nervous system | 70 | 19.6 | 62 | 17.3 | 99 | 28.0 | 71 | 20.4 | 92 | 27.7 | 98 | 29.1 | 85 | 23.2 | 74 | 20.9 | 72 | 19.7 |
| Q 10-18 Congenital malformations of eye, ear, face and neck | 25 | 7.0 | 11 | 3.1 | 17 | 4.8 | 23 | 6.6 | 19 | 5.7 | 25 | 7.4 | 18 | 5.3 | 19 | 5.4 | 11 | 3.0 |
| Q20-28 Congenital malformations of the circulatory system | 277 | 77.5 | 281 | 78.6 | 506 | 143.3 | 454 | 130.4 | 441 | 132.6 | 474 | 141.0 | 560 | 166.0 | 449 | 126.7 | 504 | 137.7 |
| Q 30-34 Congenital malformations of the respiratory system | 19 | 5.3 | 10 | 2.8 | 29 | 8.2 | 26 | 7.5 | 24 | 7.2 | 34 | 10.1 | 28 | 8.3 | 11 | 3.1 | 13 | 3.6 |
| Q35-37 Cleft lip and cleft palate | 82 | 22.9 | 52 | 14.5 | 67 | 19.0 | 66 | 19.0 | 55 | 16.5 | 64 | 19.0 | 58 | 17.2 | 62 | 17.5 | 49 | 13.4 |
| Q38-45 Congenital malformations of the digestive system | 49 | 13.7 | 39 | 10.9 | 50 | 14.2 | 49 | 14.1 | 35 | 10.5 | 48 | 14.3 | 43 | 12.7 | 47 | 13.3 | 58 | 15.8 |
| Q50-56 Congenital malformations of genital organs | 96 | 26.8 | 80 | 22.4 | 72 | 20.4 | 82 | 23.6 | 66 | 19.8 | 80 | 23.8 | 57 | 16.9 | 62 | 17.5 | 46 | 12.6 |
| Q60-64 Congenital malformations of the urinary system | 41 | 11.5 | 42 | 11.7 | 64 | 18.1 | 59 | 17.0 | 62 | 18.6 | 168 | 50.0 | 169 | 50.1 | 155 | 43.7 | 138 | 37.7 |
| Q65-79 Congenital malformations of the musculoskeletal system | 243 | 68.0 | 164 | 45.9 | 214 | 60.6 | 180 | 51.7 | 206 | 61.9 | 216 | 64.2 | 207 | 61.4 | 158 | 44.6 | 164 | 44.8 |
| Q80-87 Congenital malformations of integument | 9 | 2.5 | 4 | 1.1 | 7 | 2.0 | 66 | 1.7 | 10 | 3.0 | 8 | 2.4 | 3 | 0.9 | 4 | 1.1 | 7 | 1.9 |
| Q86-87 Other congenital malformation syndromes affecting multiple systems | 85 | 23.8 | 67 | 18.7 | 85 | 24.1 | 92 | 26.4 | 75 | 22.6 | 90 | 26.8 | 81 | 24.0 | 84 | 23.7 | 66 | 18.0 |
| Q89 Other congenital malformations, not elsewhere classified | 5 | 1.4 | 0 | 0.0 | 5 | 1.4 | 2 | 0.6 | 2 | 0.6 | 3 | 0.9 | 4 | 1.2 | 5 | 1.4 | 9 | 2.5 |
| Q 90-99Chromosomal abnormalities, not elsewhere classified | 61 | 17.1 | 44 | 12.3 | 83 | 23.5 | 82 | 23.6 | 67 | 20.6 | 78 | 23.2 | 85 | 25.2 | 75 | 21.2 | 60 | 16.4 |
| Other, not classified by Q ICD10 | | | | | 5 | 1.4 | 10 | 2.9 | 10 | 3.0 | 13 | 3.9 | 10 | 3.0 | 15 | 4.2 | 8 | 2.2 |

Table 10. Prevalence of congenital anomalies in Wielkopolska province in 2007 (on 10,000 births)

| Districts | 2007# | | |
|---|---|---|---|
| | N | LB&SB* | Prevalence |
| Powiat chodzieski | 37 | 509 | 726.9 |
| Powiat czarnkowsko-trzcianecki | 23 | 985 | 233.5 |
| Powiat gnieźnieński | 25 | 1608 | 155.5 |
| Powiat gostyński | 15 | 857 | 175.0 |
| Powiat grodziski | 11 | 619 | 177.7 |
| Powiat jarociński | 14 | 766 | 182.8 |
| Powiat kaliski | 17 | 799 | 212.8 |
| Powiat kępiński | 7 | 655 | 106.9 |
| Powiat kolski | 22 | 893 | 246.4 |
| Powiat koniński | 39 | 1408 | 277.0 |
| Powiat kościański | 11 | 811 | 135.6 |
| Powiat krotoszyński | 14 | 862 | 162.4 |
| Powiat leszczyński | 14 | 580 | 241.4 |
| Powiat międzychodzki | 11 | 412 | 267.0 |
| Powiat nowotomyski | 24 | 844 | 284.4 |
| Powiat obornicki | 18 | 668 | 269.5 |
| Powiat ostrowski | 54 | 1633 | 330.7 |
| Powiat ostrzeszowski | 13 | 602 | 215.9 |
| Powiat pilski | 125 | 1506 | 830.0 |
| Powiat pleszewski | 14 | 689 | 203.2 |
| Powiat poznański | 59 | 3384 | 1743 |
| Powiat rawicki | 14 | 694 | 201.7 |
| Powiat słupecki | 16 | 631 | 253.6 |
| Powiat szamotulski | 30 | 966 | 310.6 |
| Powiat średzki | 12 | 573 | 209.4 |
| Powiat śremski | 16 | 687 | 232.9 |
| Powiat turecki | 12 | 652 | 184.0 |
| Powiat wągrowiecki | 24 | 836 | 287.1 |
| Powiat wolsztyński | 11 | 678 | 162.2 |
| Powiat wrzesiński | 16 | 858 | 186.5 |
| Powiat złotowski | 39 | 766 | 509.1 |
| Powiat m. Kalisz | 19 | 1008 | 188.5 |
| Powiat m. Konin | 20 | 758 | 263.9 |
| Powiat m. Leszno | 16 | 636 | 251.6 |
| Powiat m. Poznań | 53 | 5577 | 95.0 |
| Total | 873 | 36610 | 238.5 |

# – temporary data
N – number of children with congenital anomalies
LB&SB* – live births and stillbirths in 2006

In that region, heart and musculoskeletal congenital defects were the most frequent ones. The 2007 data on the incidence of congenital malformations in particular districts of the Wielkopolska province are shown in Table 10. The highest incidence of congenital malformations per 10,000 births was observed in the following districts: Piła, Chodzież and Złotów.

## The incidence of infectious diseases based on the data from the Voivodeship Sanitary and Epidemiological Station in Poznań

Communicable diseases have always constituted a worldwide problem. [NIZP-PZH, GIS 2004–2008] Therefore, recognised scientific bodies from both Europe and from around the world attach a lot of importance to threats posed by such diseases, and, consequently, stress the need to monitor their occurrence and take coordinated preventive measures. Such measures should aim at both a temporary elimination of the threats and in the long-term their total eradication. [Zieliński 2006] The incidence of infectious diseases over the last several decades has forced healthcare officials to implement such preventive and mitigation measures against these diseases that would be both legal and effective. The underlying idea of such measures is the obligation to inform about incidence of infectious diseases and to enter such incidence into the registry. The registry was initiated in Poland in 1919. [Magdzik 2002] Currently, pursuant to the Polish Communicable Diseases Act of 8 December 2008, the incidence of 59 diseases needs to be entered into the registry. All communicable diseases analysed in this study are among the 59 ones covered by the registry [Dziennik Ustaw 2008].

### Pertussis

After the incidence of pertussis dropped considerably in 2006, i.e. there was a ten-fold decrease in the number of pertussis cases in comparison with the year 2004, it was back on the increase and there was a shift towards the groups of older children (10–14 years). In the year 2008 the incidence increased by more than 300% in comparison with the year 2006. In the year 2005 the recorded cases dropped by nearly 44% in comparison with the year 2004 [NIZP-PZH, GIS 2004–2008] (see Fig. 1).

### Measles

Over the period discussed in this study, isolated cases in the Wielkopolska province were recorded in the years 2006 and 2008. The incidence of measles in children and adolescents accounted for as little as 25% of all cases and it concerned mainly two groups – aged 0–4 and 10–14 [NIZP-PZH, GIS 2004–2008] (see Fig. 2).

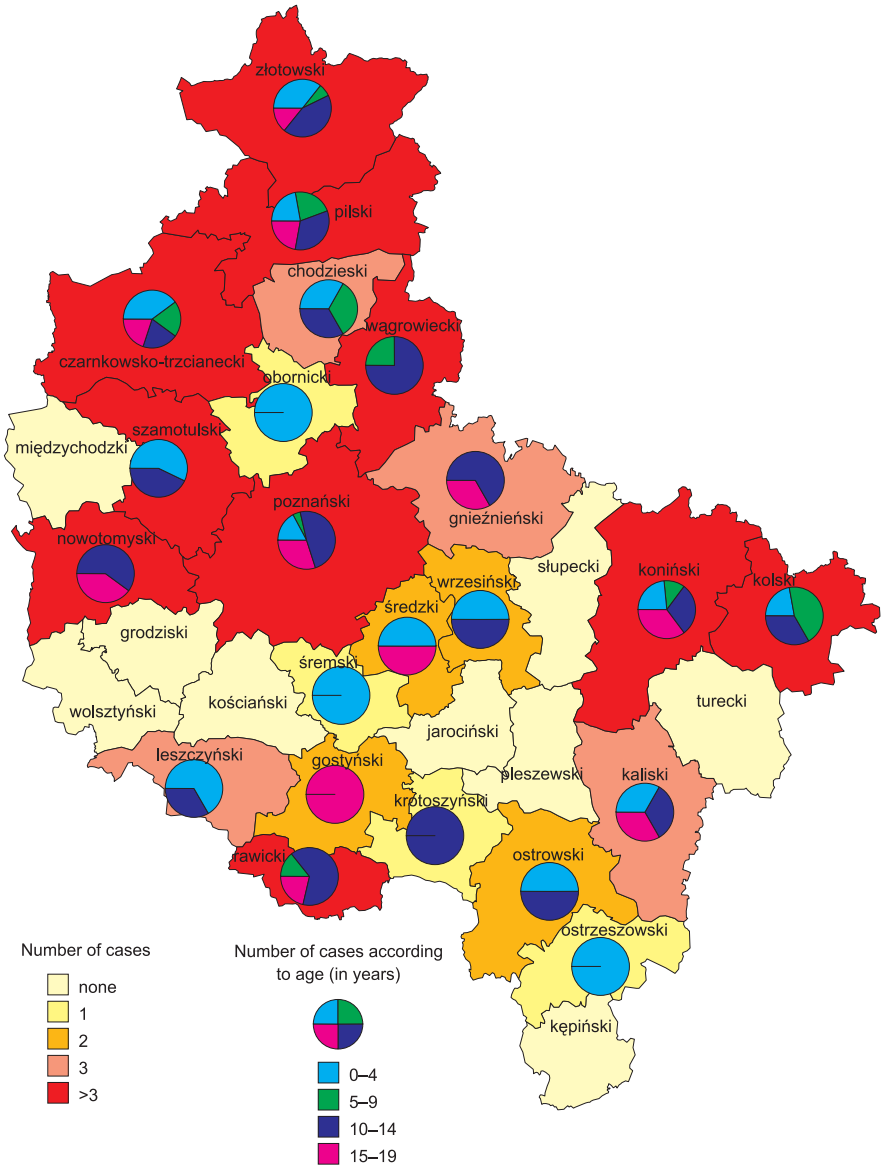



Fig. 1. Pertussis 2006–2008 – number of cases according to age

## Mumps

The incidence of mumps decreased considerably. In 2005 the number of recorded cases fell by 30% in comparison with the year 2004. In 2006 the number of recorded cases fell even more, i.e. by 80%, but in 2007 there was a record, i.e. a seven-fold decrease. Such a record decrease in the number of cases was, most probably, the effect of the introduction of obligatory vaccination (MMR vaccine). The

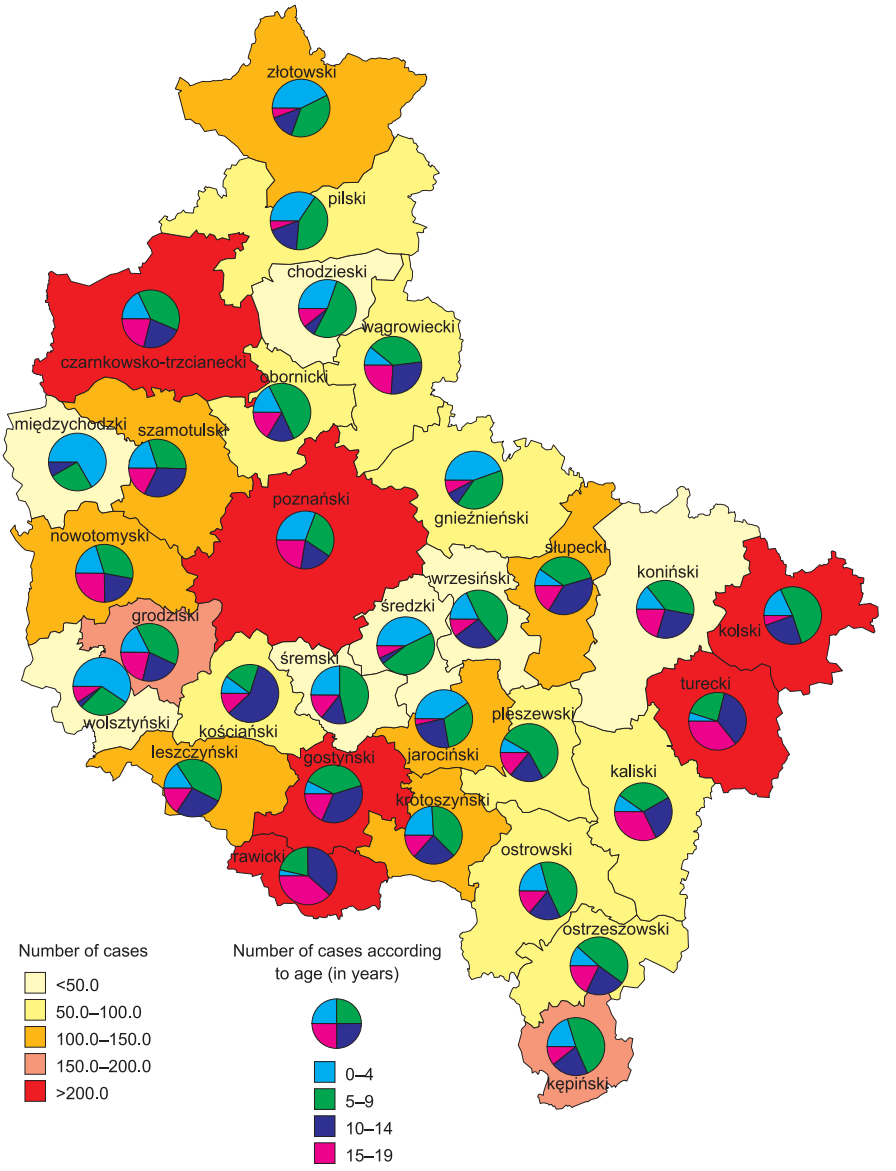



Fig. 2. Measles – cases according to age in the province of Wielkopolska in years 2006–2008

highest incidence of mumps was found among children aged 5–9 [NIZP-PZH, GIS 2004–2008] (see Fig. 3).

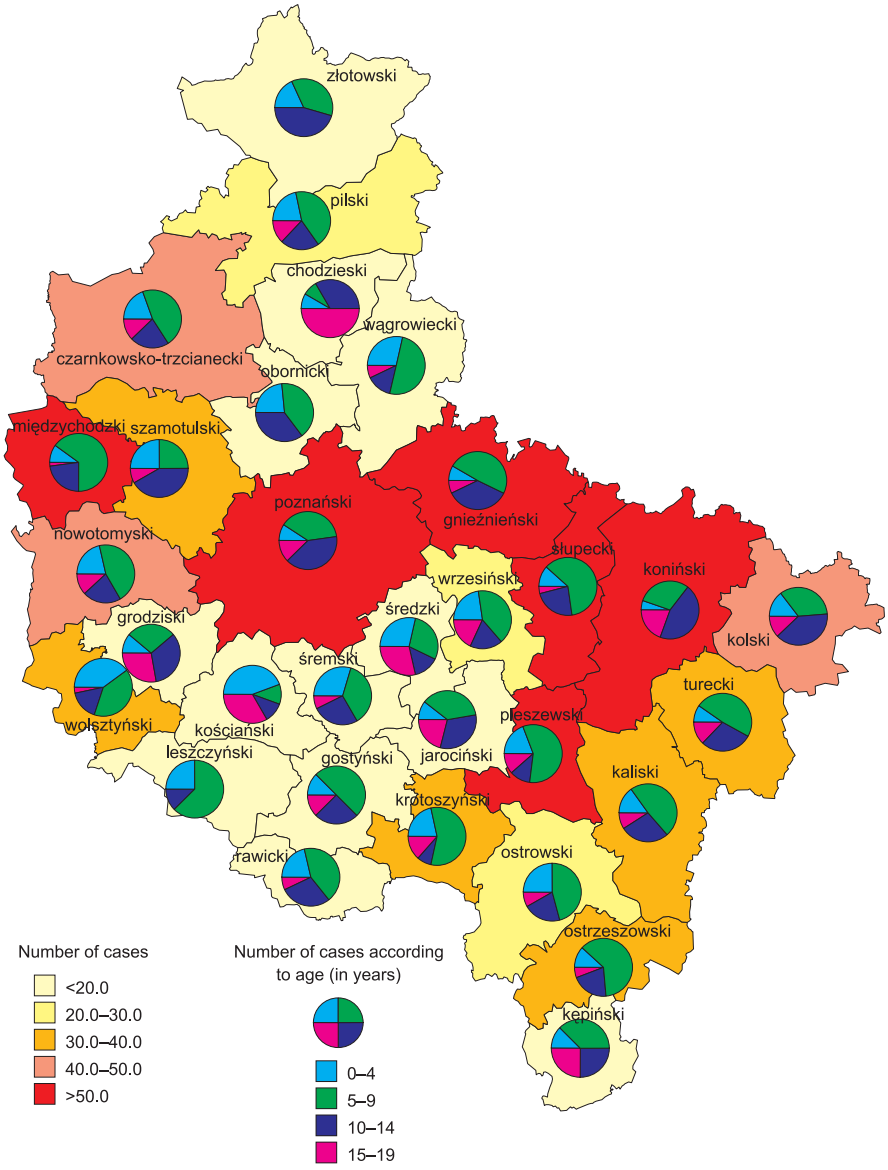



Fig. 3. Mumps 2006–2008 – number of cases according to age

## Scarlet fever

There was an increase in the incidence of scarlet fever since 2004, with the highest incidence among groups aged 0–4 and 5–9. This comes as no surprise as the epidemic cycle of scarlet fever and the periods of the increased incidence of the disease related to the cycle, take place every 7–9 years on average [NIZP-PZH, GIS 2004–2008] (see Fig. 4).

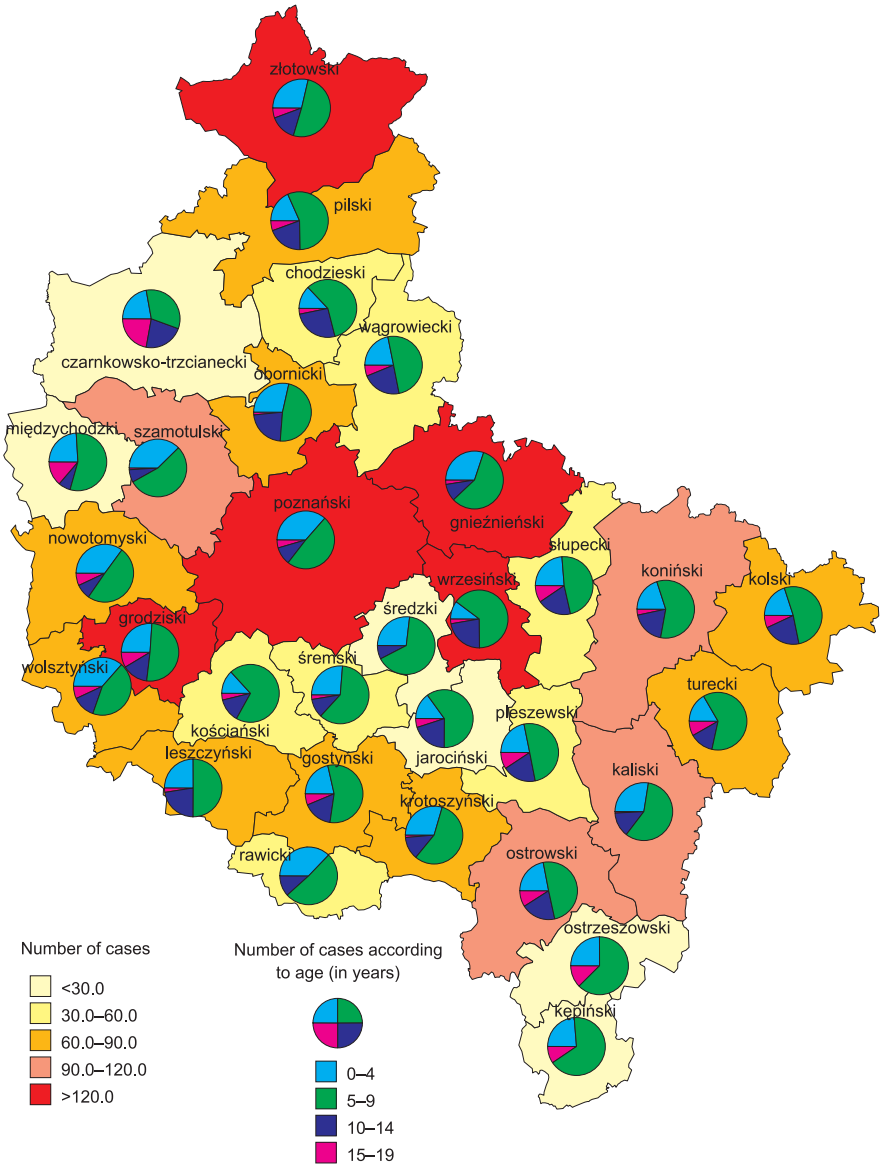



Fig. 4. Scarlet fever 2006–2008 – number of cases according to age

## Tuberculosis

The Wielkopolska province is not at a particular risk of high incidence of tuberculosis. Nevertheless, in the discussed period of 2005–2008 there was an increase in the newly recorded cases among children and adolescents, though, in general, new cases are rarely recorded in the province [NIZP-PZH, GIS 2004–2008] (see Fig. 5).

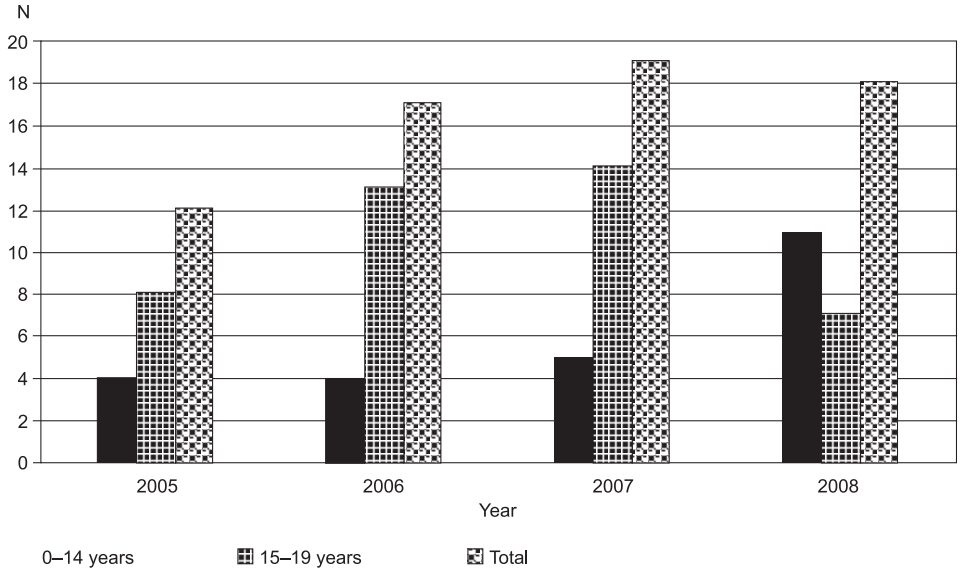



Fig. 5. Tuberculosis 2006–2008 – number of cases according to age

## Influenza

Influenza or suspicion of influenza continue to be the most frequent contagious disease (with the exception of diarrhoeas in children up to 2 years old). As data representative of the entire population of the Wielkopolska province show, after the 2005 peak in the incidence of the disease, a fall in the number of new cases was being observed until 2008. An increase was observed in children and adolescents aged 0–14. There was much fluctuation in the incidence of influenza across districts. The incidence was relatively high over the period discussed in the following districts: Kępno, Koło, Poznań, and Rawicz; whereas in the Chodzież, Jarocin, and Pleszew districts only isolated cases were observed [NIZP-PZH, GIS 2004–2008] (see Fig. 6).

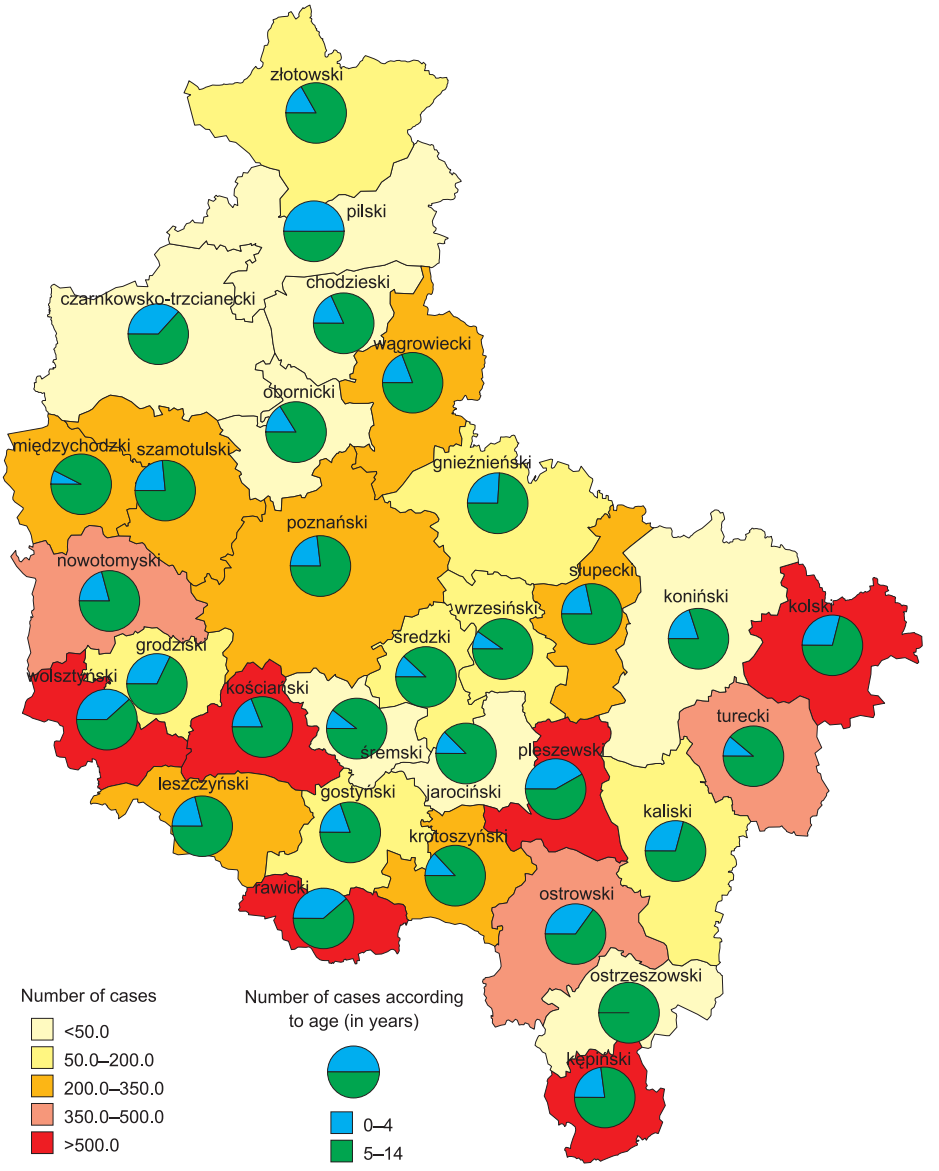



Fig. 6. Influenza 2006–2008 – number of cases according to age

## Rubella

In the Wielkopolska province, the number of cases increased from 518 in 2004 up to 1,647 in 2008. Among children and adolescents, most frequently those aged 5–9 contracted rubella, which is disturbing as in 2004 obligatory vaccination (MMR-II vaccine) against rubella was introduced. That should have theoretically resulted in a systematic fall in new cases [NIZP-PZH, GIS 2004–2008] (see Fig. 7).

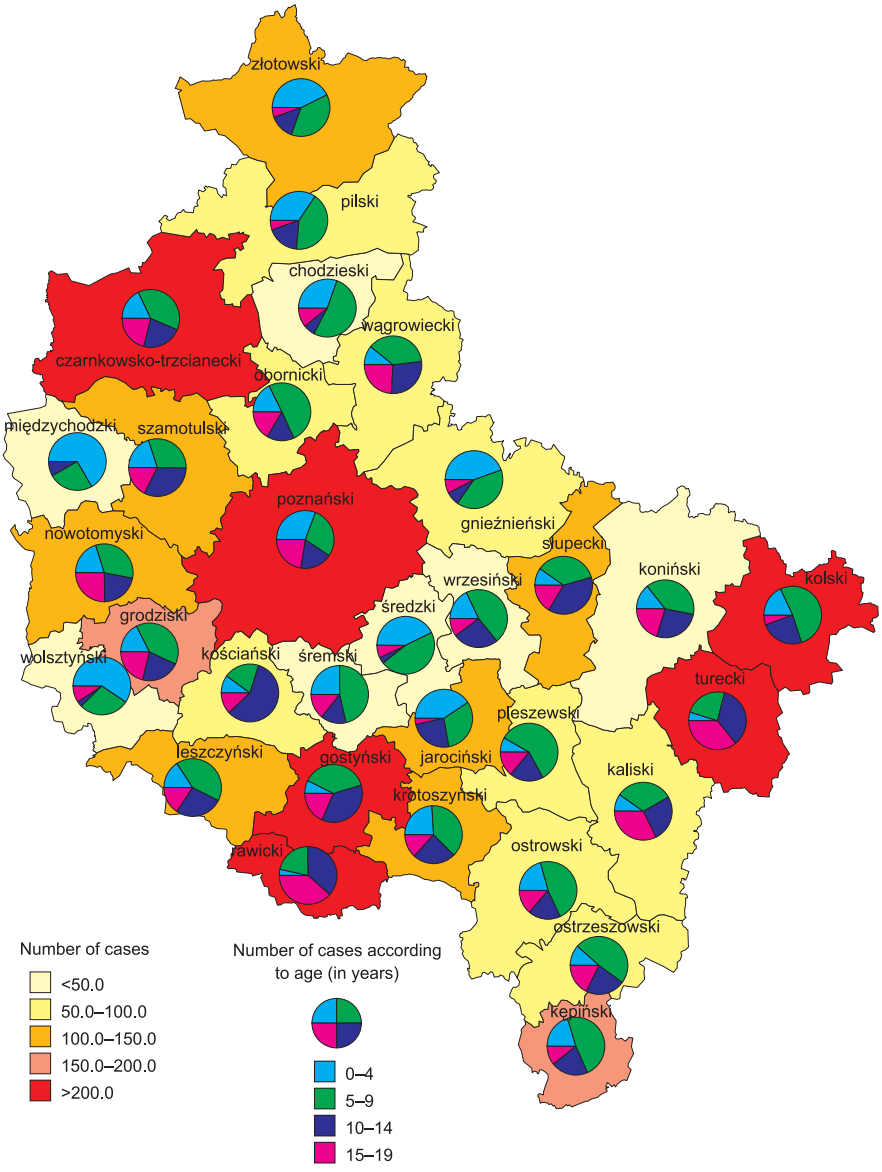



Fig. 7. German measles 2006–2008 – number of cases according to age

## Chicken pox

In the period discussed in this study there are no discernible trends. The incidence ranged from 18,395 in 2005 to 12,925 in 2008. Among children, the highest incidence was among those aged 0–4 and 5–9. Vaccination against chicken pox has been recommended for four years now, which may in the long term lead to a considerable drop in the incidence of the disease [NIZP-PZH, GIS 2004–2008] (see Fig. 8).

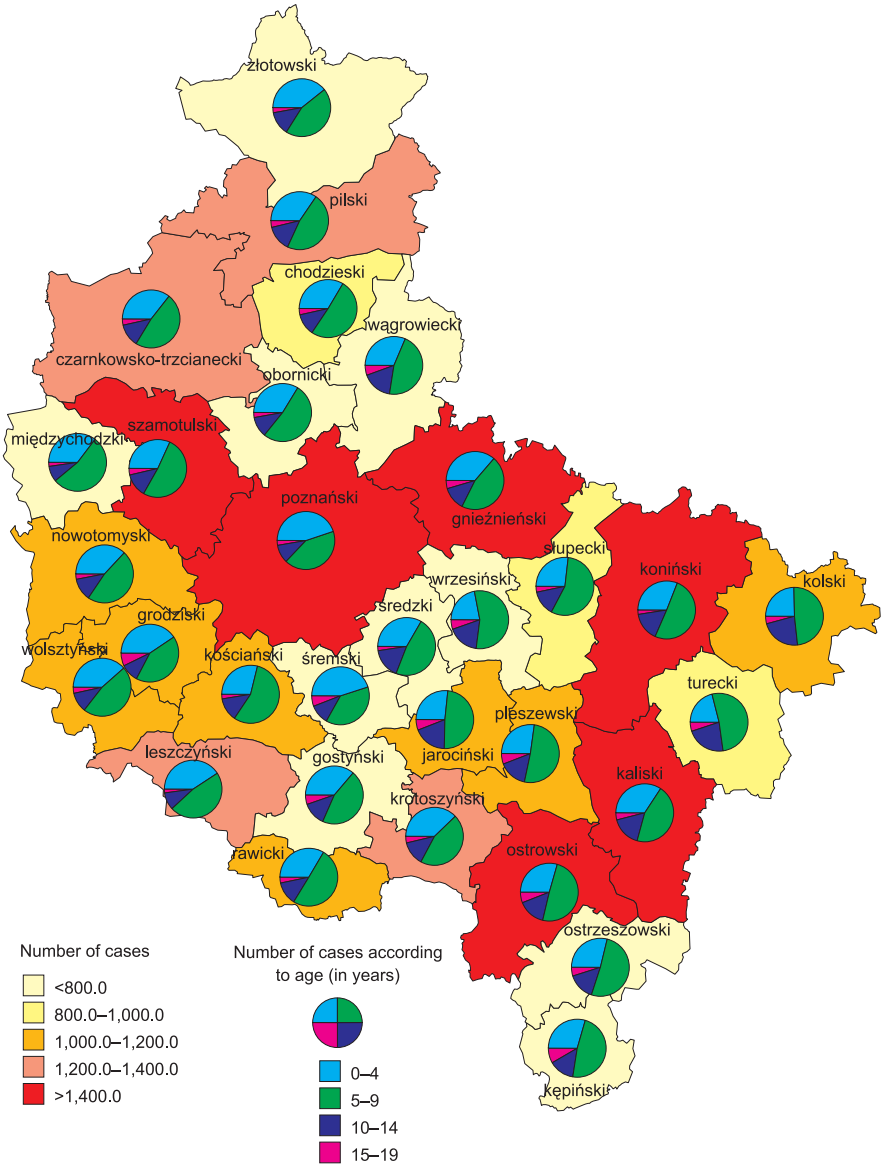



Fig. 8. Chicken pox 2006–2008 – number of cases according to age

## Meningococcal infections

The incidence of invasive meningococcal infections in Poland in 2008 was 0.97 per 100,000 people, which means that there were 369 cases in the entire country in 2008, and at 1.06 per 100,000 people in the Wielkopolska province with 36 cases. In the years 2004–2008 a continuous increase in the incidence of meningococcal infections in the Wielkopolska province was observed (9 cases in 2004 and 36 in 2008). A continuous increase in the incidence was also observed among children (in 2008 there was an increase by 150% in comparison with the year 2006). The largest number of cases was observed among the youngest children (aged 0–4) and adolescents (aged 15–19) [NIZP-PZH, GIS 2004–2008] (see Fig. 9).

## Salmonellosis

In the Wielkopolska province, the number of food poisonings and parenteral infections caused by salmonella bacilli remained relatively stable, ranging from 1205 cases in 2004 to 971 in 2006. There was, however, a fall in the incidence among children and adolescents. Among them, the highest incidence was in the youngest children (0–4), but, at the same time, in 2008 the incidence fell by 25% in comparison with 2006. What is notable is the fact that a relatively high incidence was consistent in some districts, such as: Gniezno, Kępno, Konin, Leszno and Poznań [NIZP-PZH, GIS 2004–2008] (see Fig. 10).

## Hepatitis

Until the end of 1980s, the epidemiological status of hepadnavirus infections in Poland had been one of the most serious ones across Europe. Since 1992 there has been a systematic decrease in the incidence of HBV infections. In 2002 the incidence was 5.3 cases per 100,000 people, in 2003 – at 4.7 per 100,000, and in 2006 – 4.4 per 100,000.

In the years 2004–2008 a stable and relatively low incidence of hepatitis B in the Wielkopolska province was accompanied by a drop in hepatitis A, on the one hand, and an increase in hepatitis C, on the other. Since 2004 up till now, there have been considerably more cases of hepatitis C than hepatitis B [NIZP-PZH, GIS 2004–2008].

### Hepatitis A

In the period analysed, there were only isolated cases of hepatitis A recorded in the entire population as well as among children and adolescents.

### Hepatitis B

The highest incidence of hepatitis B was recorded in 2007. In the period discussed in this study the highest incidence was observed for groups aged 10–14 and 15–19 (see Fig. 11).

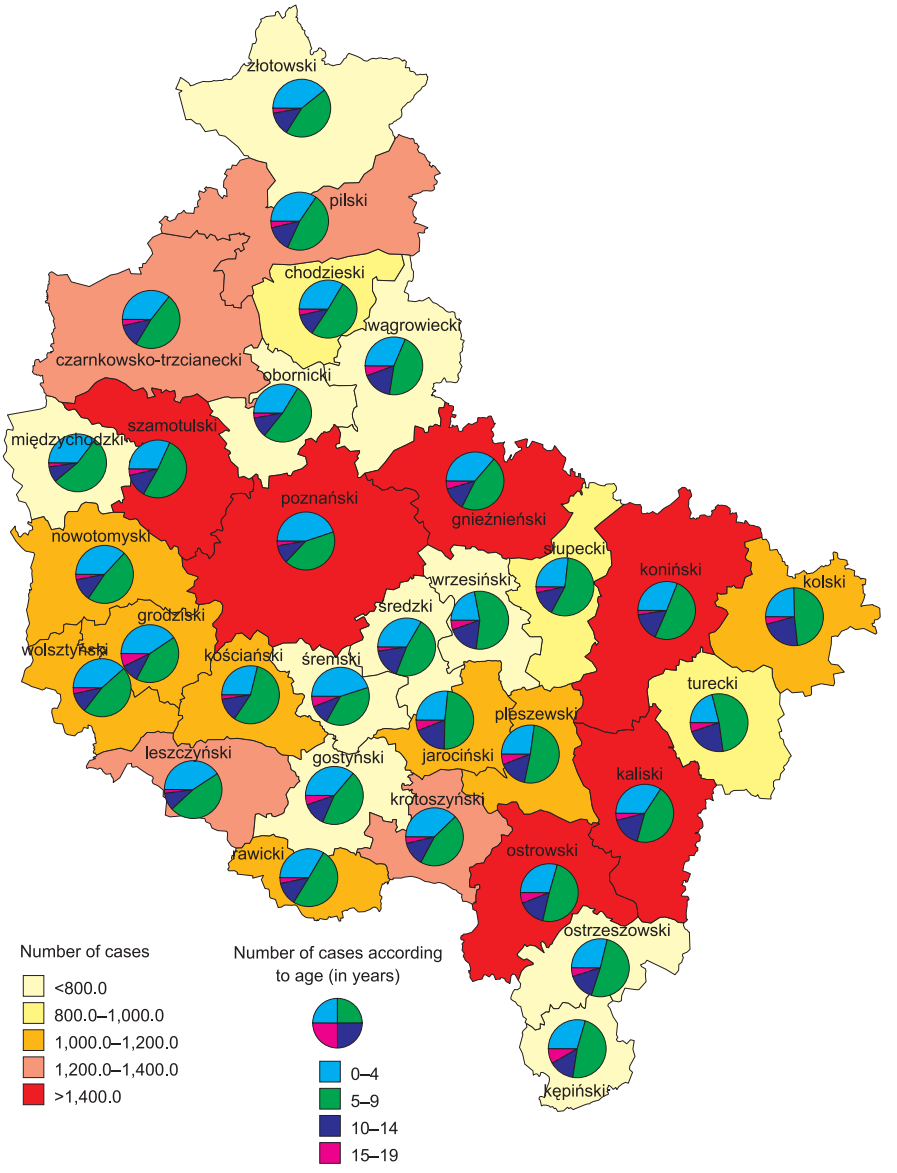



Fig. 9. Meningococcal infections 2006–2008 – number of cases according to age

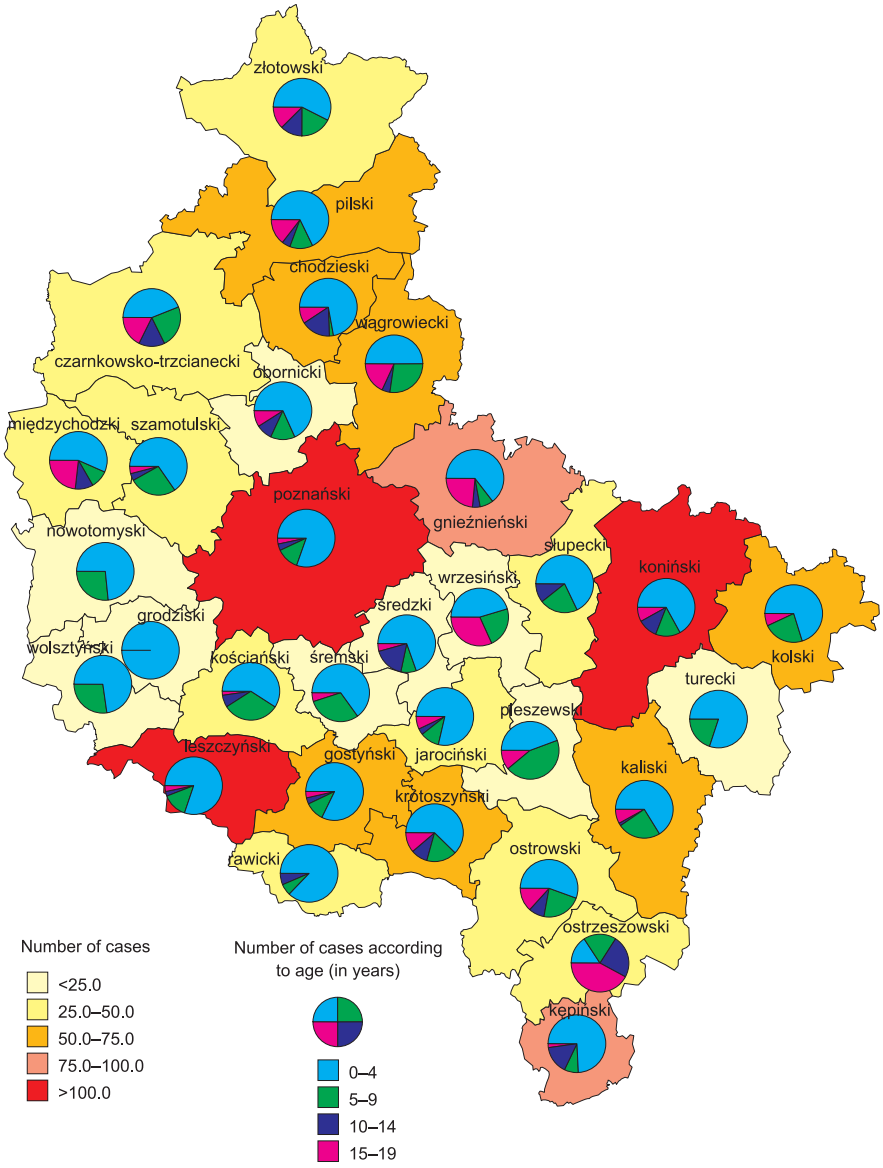



Fig. 10. Salmonellosis 2006–2008 – number of cases according to age

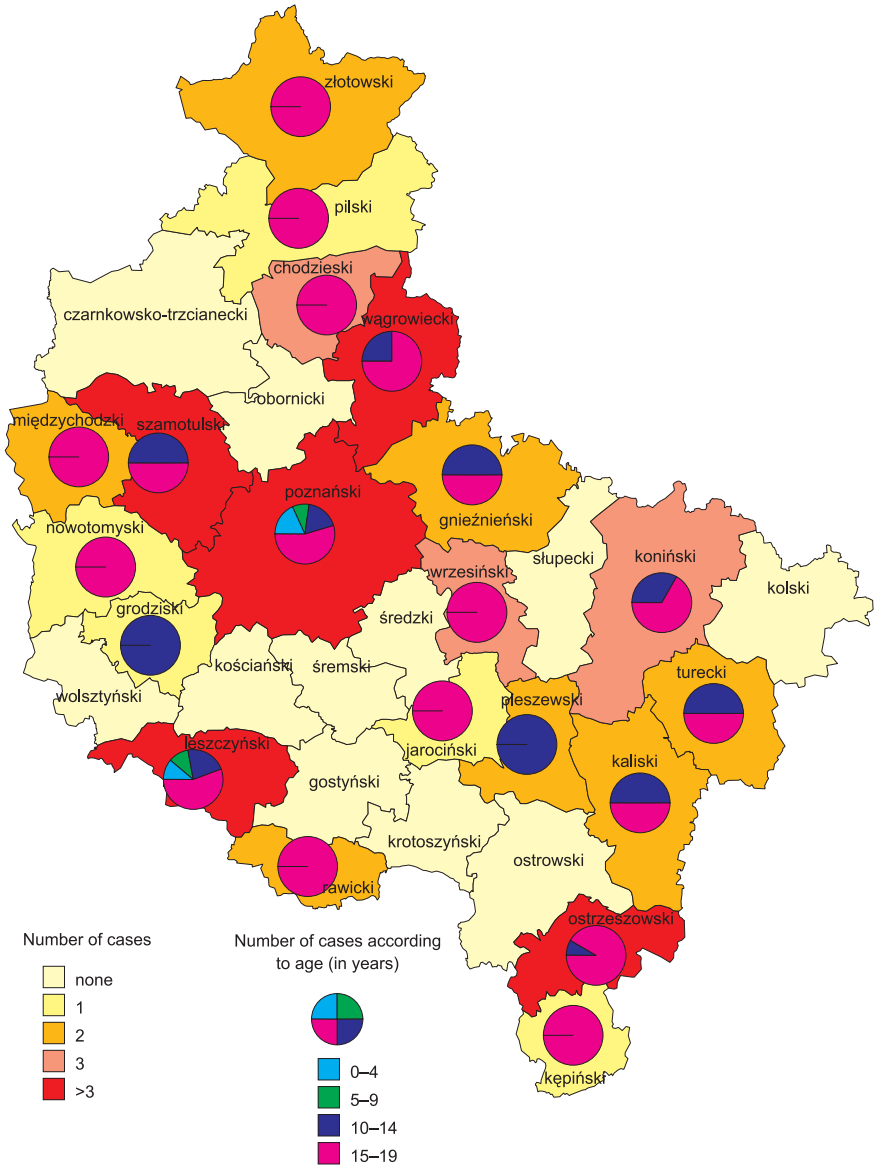



Fig. 11. Hepatitis B 2006–2008 – number of cases according to age

## Hepatitis C

Hepatitis C proves to be the most significant problem. In the years analysed, the number of cases nearly doubled since 2004. There was much discrepancy between the incidence among the youngest (0–19) and the rest of the population, as only about 10% of cases concerned the youngest. Within that group those aged 15–19 were most prone to contract the infection. Interestingly, the disease was prevalent in specific locations during all years analysed (see Fig. 12).

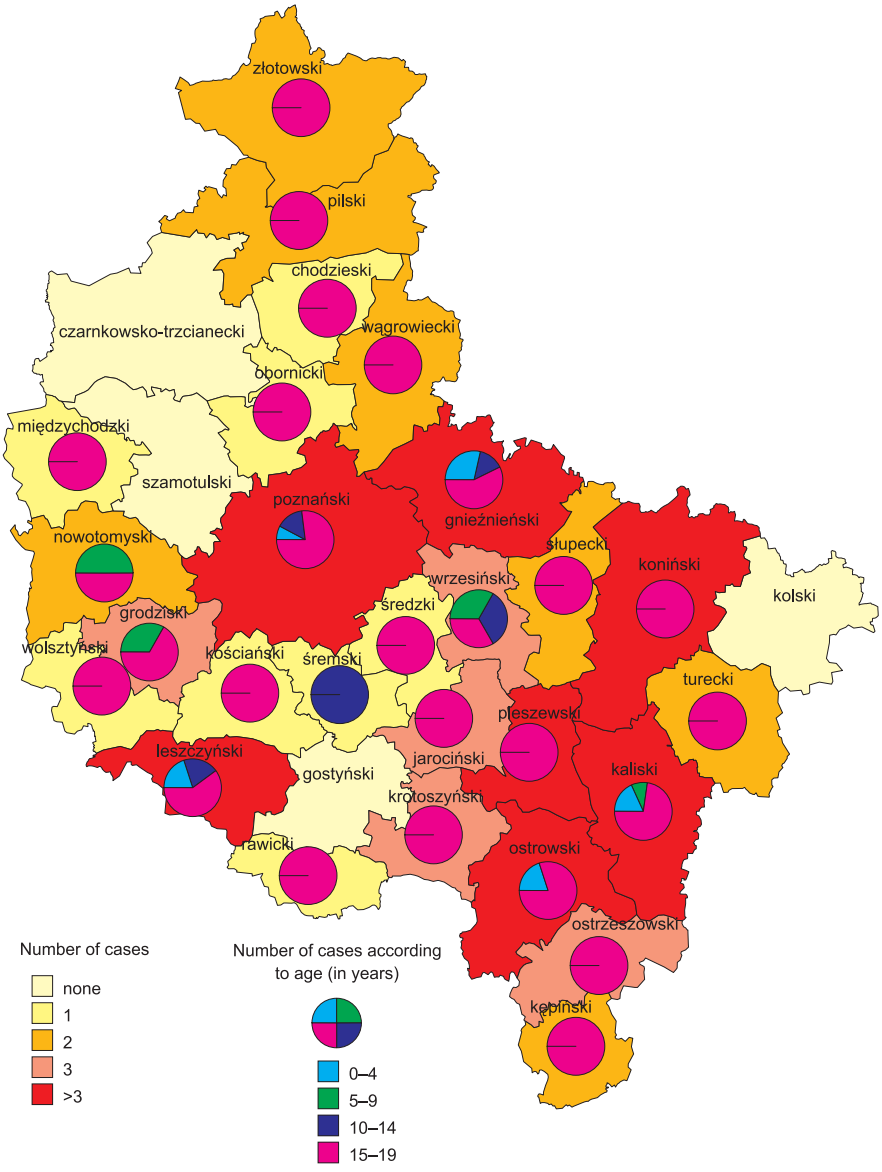



Fig. 12. Hepatitis C 2006–2008 – number of cases according to age

**Neoplasms in children and adolescents aged 0–19 in the Wielkopolska province in the years 2004–2007.**

Extremely high incidence of particular kinds of neoplasms is specific to the Wielkopolska province. Therefore, physicians need to be highly vigilant and need to intensify their activities in the area of prevention, early diagnosis and treatment not only among the adult population, but also among children and adolescents. The data from the Wielkopolska Regional Centre for Public Health shows that neoplasms were second, after external factors, cause of deaths among those aged 10–19. Regional patterns of tumour incidence in the Wielkopolska province may

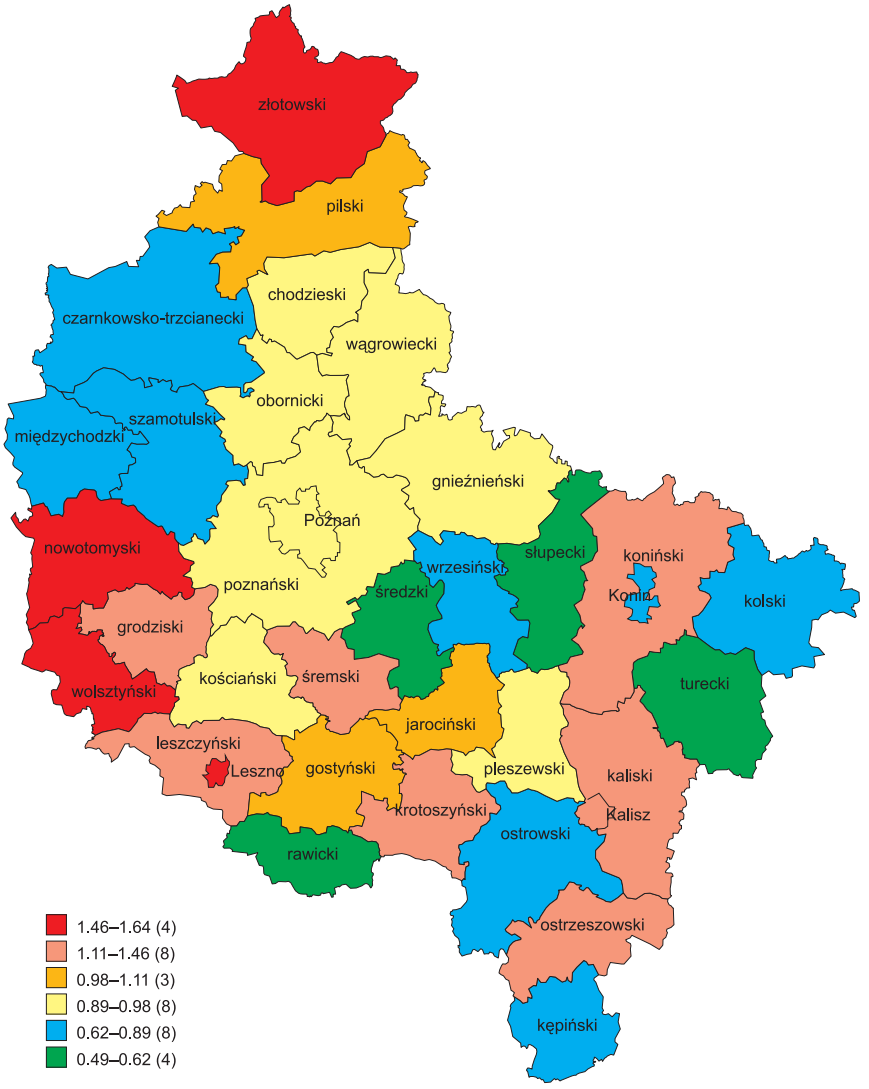



Fig. 13. The SIR model of incidence of malignant neoplasms in men in Wielkopolska province

be illustrated by means of crude incidence rate or standardized incidence rate (the SIR model). The SIR model of incidence of malignant tumour in men in the Wielkopolska province in the years 1999–2006 is presented in Fig. 13.

The equation SIR=1 means that the incidence in the relevant district was similar to that in the Wielkopolska province. SIR>1 suggests that the incidence in the relevant district was higher to that in the Wielkopolska province, whereas SIR<1 suggests that the incidence in the relevant district was lower to that in the Wielkopolska province. The highest incidence of malignant tumour in men was observed in northern part of the province and in the Śrem district. The lowest incidence was found in the following districts: Ostrzeszów, Wolsztyn, and Grodzisk. The highest incidence among women in the years 1999–2006 was found in Szamotuły, Obroniki, Chodzież, and Jarocin districts, as well as urban districts of Leszno and Konin. The lowest incidence was found in eastern districts (with the exception of urban districts) as well as Wolsztyn and Grodzisk districts (see Fig. 14).

The number of neoplasm among boys aged 0–19 ranged from 60 in the year 2005 to 73 in 2006. In each of the discussed years, the highest incidence was in group aged 15–19 (90 cases in total in the years 2004–2007), followed by group aged 0–4 (70 cases). In the latter group cases of lymphatic leukaemia as well as of malignant brain and kidney tumours were the most prevalent ones (Table 11). Among boys aged 15–19 the most frequent cases were those of: malignant testicle tumour, Hodgkin's disease, lymphatic leukaemia, as well as malignant brain, bone and cartilage tumours (Table 11). The incidence of malignant tumours among girls aged 0–19 was highest in 2004 (61 cases) and lowest in 2007 (43 cases). Just like in boys, the highest incidence among girls was in group aged 15–19 (87 cases in total in the years 2004–2007), followed by group aged 0–4 (54 cases) (Table 12). Among girls aged 15–19 cases of Hodgkin's disease were the most frequent ones, followed by malignant brain and thyroid tumours. Among the youngest, i.e. those aged 0–4, lymphatic leukaemia and malignant brain tumour cases were the most prevalent ones.

In the 2004–2007 the distribution of malignant tumour incidence in boys aged 0–19 in the Wielkopolska province, lymphatic leukaemia cases (C91-C95) were the most prevalent ones in each year, accounting for 20.5–39.3% of total incidence. In the years 2004–2006 malignant brain and central nervous system tumours were second most frequent types. Hodgkin's disease was ranked third in 2004 and 2005. Over the last two years of the study there was a shift in the incidence of malignant testicle tumour (ranked third in 2006, and second in 2007). The structure of the malignant tumour incidence in boys aged 0–19 in Poland in the years 2004–2007 proved to be more uniform. Leukaemia accounted for the biggest proportion of cases (24.1%–26.5%). Over the same period of time malignant brain and nervous system tumours ranked second (17.9%–19.0%), whereas Hodgkin's disease was third in the years 2005–2007.

The 2004–2007 distribution of malignant tumour incidence in girls aged 0–19 proved to be similar to that observed in the Wielkopolska province. Here, in the years 2005–2007 leukaemias also accounted for the biggest proportion of cases, followed by malignant brain and nervous system tumours and Hodgkin's disease.

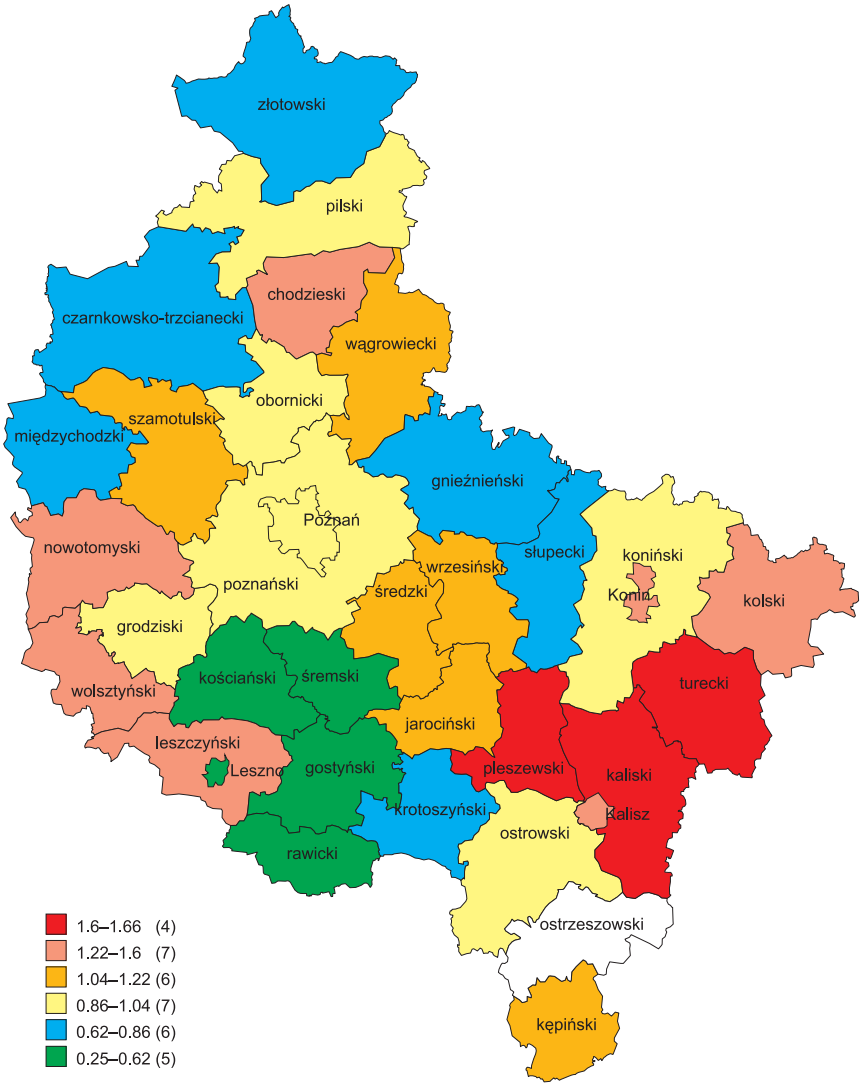



Fig. 14. The SIR model of incidence of malignant neoplasms in women in Wielkopolska province

The incidence of malignant brain and nervous system tumours was higher than that of leukaemia only in 2004, accounting for 32.0% of all cases at that time.

The distribution of the malignant tumours in boys and girls aged 0–19 in the Wielkopolska province and in Poland in 2007 is presented in Fig. 15, 16, 17 and 18.

Table 11. Number of cases of malignant neoplasms in boys aged 0–19 in Wielkopolska province in 2004–2007

| Type of neoplasms or group of malignant neoplasms | 2004 | | | | 2005 | | | | 2006 | | | | 2007 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Age/Years | | | | | | | | | | | | | | | |
| | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 |
| Malignant neoplasm of parotid gland(C07) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| Malignant neoplasm of nasopharynx(C11) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Malignant neoplasm of liver (C22) | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of nasal cavity and middle ear (C30) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of accessory sinuses (C31) | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of heart, mediastinum and pleura (C38) | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of bone and articular cartilage of limbs (C40) | 0 | 1 | 0 | 2 | 0 | 0 | 1 | 3 | 0 | 0 | 0 | 3 | 1 | 0 | 2 | 1 |
| Malignant neoplasm of bone and articular cartilage of other and unspecified sites (C41) | 1 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 |
| Malignant melanoma of skin (C43) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Other malignant neoplasms of skin (C44) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of peripheral nerves and autonomic nervous system (C47) | 0 | 0 | 0 | 0 | 2 | 1 | 1 | 0 | 2 | 1 | 0 | 0 | 1 | 0 | 0 | 0 |
| Malignant neoplasm of other connective and soft tissue (C49) | 1 | 1 | 1 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 1 | 0 |
| Malignant neoplasm of testis (C62) | 0 | 0 | 0 | 6 | 0 | 0 | 0 | 4 | 1 | 0 | 0 | 7 | 0 | 0 | 0 | 7 |
| Malignant neoplasm of other and unspecified male genital organs (C63) | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

| Type of neoplasms or group of malignant neoplasms | 2004 | | | | 2005 | | | | 2006 | | | | 2007 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Age/Years | | | | | | | | | | | | | | | |
| | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 |
| Malignant neoplasm of kidney (C64) | 4 | 2 | 0 | 0 | 1 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| Malignant neoplasm of bladder (C67) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of eye and adnexa (C69) | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 0 | 0 | 0 | 2 | 0 | 0 | 0 |
| Malignant neoplasm of brain (C71) | 2 | 5 | 2 | 3 | 3 | 1 | 2 | 3 | 4 | 2 | 2 | 4 | 1 | 1 | 1 | 1 |
| Malignant neoplasm of spinal cord, cranial nerves and other parts of CNS ( C72) | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of thyroid gland (C73) | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of adrenal gland (C74) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 0 | 0 | 2 | 0 | 0 | 0 |
| Malignant neoplasm of other endocrine glands and related structures (C75) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Hodgkin's disease (C81) | 0 | 1 | 2 | 4 | 0 | 2 | 1 | 3 | 1 | 1 | 1 | 2 | 0 | 0 | 0 | 4 |
| Diffuse non–Hodgkin's lymphoma (C83) | 0 | 2 | 0 | 2 | 1 | 1 | 1 | 0 | 0 | 1 | 3 | 1 | 0 | 4 | 0 | 0 |
| Peripheral and cutaneous T-cell lymphomas (C84) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Other and unspecified types of non–Hodgkin's lymphoma (C85) | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 |
| aautoLymphoid leukaemia (C91) | 5 | 3 | 1 | 4 | 4 | 2 | 1 | 4 | 5 | 5 | 1 | 0 | 5 | 2 | 4 | 4 |
| Myeloid leukaemia (C92) | 3 | 2 | 1 | 0 | 0 | 3 | 3 | 1 | 0 | 0 | 4 | 0 | 2 | 2 | 2 | 1 |
| Leukaemia of unspecified cell type (C95) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Other and unspecified malignant neoplasms of lymphoid, haematopoietic and related tissue (C96) | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 |
| Total: | 18 | 17 | 8 | 24 | 14 | 11 | 14 | 21 | 22 | 17 | 11 | 23 | 16 | 11 | 12 | 22 |

Table 12. Number of cases of malignant neoplasms in girls aged 0–19 in Wielkopolska province in 2004–2007

| Type of neoplasm or group of malignant neoplasms | 2004 | | | | 2005 | | | | 2006 | | | | 2007 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Age/Years | | | | | | | | | | | | | | | |
| | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 |
| Malignant neoplasm of parotid gland(C07) | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of liver (C22) | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of nasal cavity and middle ear (C30) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Malignant neoplasm lungs (C34) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Malignant neoplasm of heart, mediastinum and pleura (C38) | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of bone and articular cartilage of limbs (C40) | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of bone and articular cartilage of other and unspecified sites (C41) | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 1 |
| Malignant melanoma of skin (C43) | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Other malignant neoplasms of skin (C44) | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| Malignant neoplasm of peripheral nerves and autonomic nervous system (C47) | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| Malignant neoplasm of other connective and soft tissue (C49) | 0 | 1 | 2 | 1 | 1 | 0 | 0 | 1 | 1 | 1 | 0 | 2 | 2 | 0 | 1 | 0 |
| Malignant neoplasm of vagina (C52) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of uterus, part unspecified (C55) | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

| Type of neoplasm or group of malignant neoplasms | 2004 | | | | 2005 | | | | 2006 | | | | 2007 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Age/Years | | | | | | | | | | | | | | | |
| | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 | 0–4 | 5–9 | 10–14 | 15–19 |
| Malignant neoplasm of ovary (C56) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of kidney (C64) | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 1 | 1 | 2 | 0 | 0 | 1 | 0 | 0 | 0 |
| Malignant neoplasm of eye and adnexa (C69) | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Malignant neoplasm of brain (C71) | 2 | 4 | 4 | 1 | 6 | 1 | 5 | 4 | 0 | 5 | 0 | 4 | 2 | 2 | 1 | 3 |
| Malignant neoplasm of spinal cord, cranial nerves and other parts of CNS ( C72) | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of thyroid gland (C73) | 0 | 1 | 2 | 3 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 4 | 0 | 0 | 1 | 2 |
| Malignant neoplasm of adrenal gland (C74) | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Malignant neoplasm of other and ill-defined sites (C76) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Hodgkin's disease (C81) | 0 | 0 | 0 | 1 | 11 | 0 | 0 | 0 | 4 | 0 | 2 | 6 | 0 | 0 | 1 | 5 |
| Diffuse non-Hodgkin's lymphoma (C83) | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 1 | 0 |
| Other and unspecified types of non-Hodgkin's lymphoma (C85) | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Lymphoid leukaemia (C91) | 7 | 0 | 2 | 1 | 4 | 4 | 2 | 0 | 9 | 1 | 0 | 2 | 3 | 5 | 0 | 2 |
| Myeloid leukaemia (C92) | 0 | 2 | 1 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 2 | 0 | 1 |
| Leukaemia of unspecified cell type (C95) | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Total | 13 | 9 | 15 | 24 | 15 | 6 | 11 | 18 | 17 | 10 | 5 | 28 | 9 | 10 | 7 | 17 |

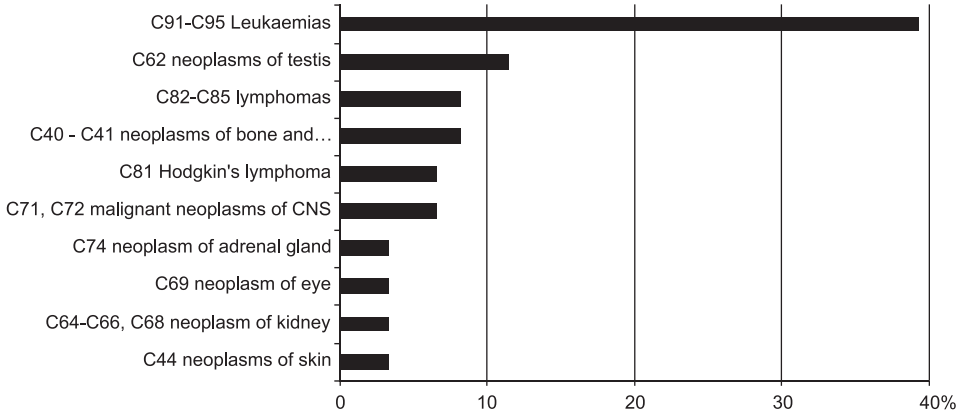



Fig. 15. Malignant neoplasms in boys (0–19) in Wielkopolska province in 2007

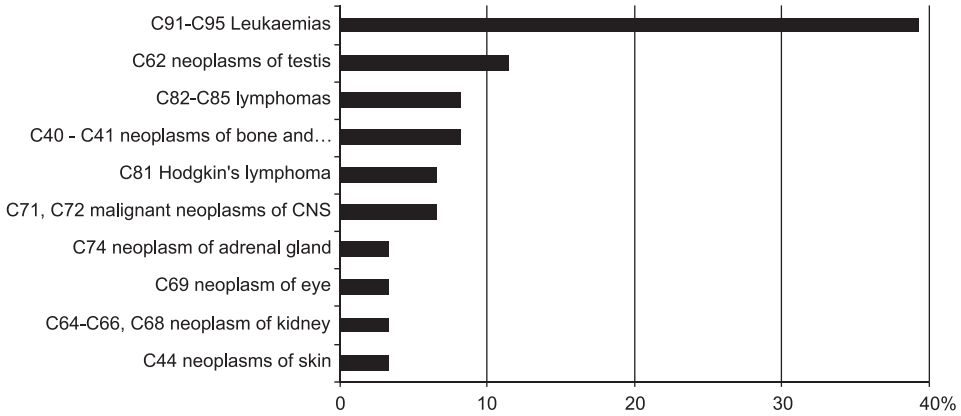



Fig. 16. Malignant neoplasms in boys (age 0–19) in Poland in 2007

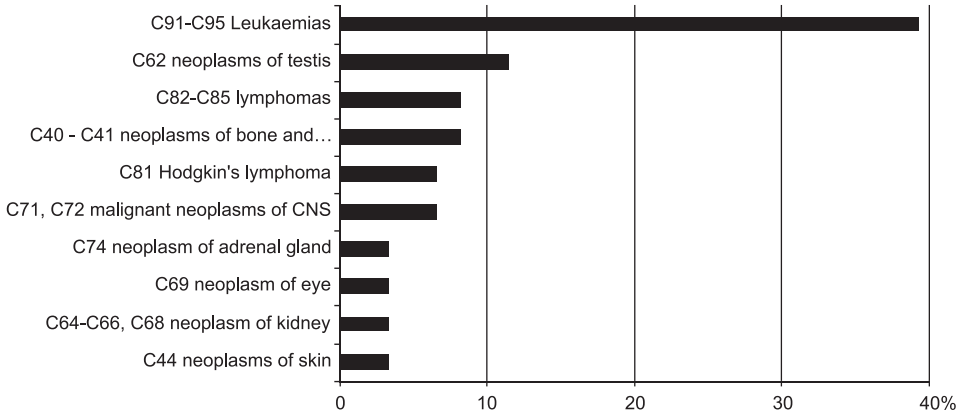



Fig. 17. Malignant neoplasms in girls (0–19) in Wielkopolska province in 2007

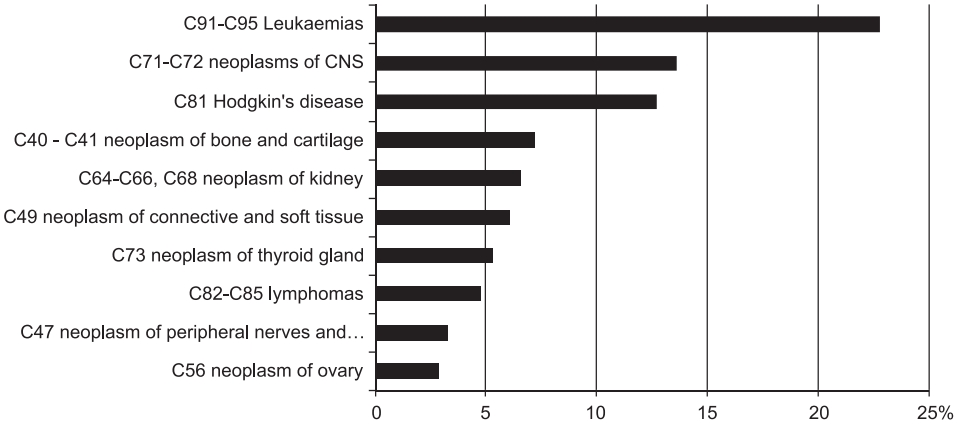


Fig. 18. Malignant neoplasms in girls (age 0–19) in Poland in 2007

## Conclusion

1. Accidents and injuries were the most frequent causes of death among adolescents.
2. Allergies as well as permanent musculoskeletal disorders and deforming dorsopathies were the most frequently recorded health problems among children and adolescents.
3. Cardiac defects and musculoskeletal congenital malformations were the most frequent congenital health problems. It should be expected that there will be an increase in the number surgical repairs of such defects among infants.
4. The implementation of the obligatory vaccination programme resulted in a significant decrease in the incidence of infections among children and adolescents.
5. Leukaemia as well as malignant brain and central nervous system tumours accounted for the biggest proportion of malignant tumour cases in children and adolescents.
6. There is a need for a more standardized method of collecting statistical data concerning the health status in the examined group.

## References

*Choroby zakaźne i zatrucia w Polsce*. NIZP-PZH, GIS, Warszawa. Roczniki 2004–2008.
Dyzmann-Sroka A. et al. (eds): *Nowotwory złośliwe w Wielkopolsce w latach 2004–2007*. Wielkopolskie Centrum Onkologii, 2006, 2007, 2008, 2009.
Godlewski D., Wojtyś P. (Eds.): *Nowotwory złośliwe w Wielkopolsce*. Ośrodek Profilaktyki i Epidemiologii Nowotworów, Poznań 2009.
Jędrychowski W.: *Epidemiologia – wprowadzenie i metody badań*. Warszawa: 1999. Wyd. Lek. PZWL.

Juszczyk J.: Globalne strategie zapobiegania chorobom zakaźnym na przełomie drugiego i trzeciego tysiąclecia: oczekiwania a rzeczywistość. *Przegl Epidemiol* 2004; 58:5–9.

Magdzik W.: *Biologiczne zagrożenia bezpieczeństwa kraju – ryzyko zakażenia szczególnie niebezpiecznymi patogenami*. Materiały konferencyjne Warszawa 2002, 3–16.

Ustawa z dnia 5 grudnia 2008 r. o zapobieganiu oraz zwalczaniu zakażeń i chorób zakaźnych u ludzi (Dz.U. z 2008 r. nr 234 poz. 1570).

Wojciechowska U., Didkowska J., Zatoński W.: *Nowotwory złośliwe w Polsce w latach 2004–2007*. Centrum Onkologii – Instytut im. M. Skłodowskiej-Curie, Warszawa 2006, 2007, 2008, 2009.

Zieliński A.: Znaczenie chorób zakaźnych w problematyce zdrowia publicznego w zjednoczonej Europie. *Przegl Epidemiol* 2006; 60:857–859.

Aldona Siwińska, Mirosława Godynicka, Alicja Krzyżaniak,
Małgorzata Krzywińska-Wiewiorowska,
Barbara Stawińska-Witoszyńska, Maria Kaczmarek

# Chronic health problems in adolescents with emphasis on cardiovascular diseases

**Abstract:** Early recognition of developmental defects, diseases and disorders is a key to effective child and adolescent health care. It enables the implementation of preventive actions and adequate treatment. Knowledge on the actual health status of children and adolescents and the conditions behind it forms the groundwork for setting priorities and undertaking structured actions in the area of preventive medicine and health promotion in this population. In the ADOPOLNOR project, a randomised population of 4,904 children and adolescents aged 10–18, students of primary and secondary schools from the Wielkopolska region, was subjected to an epidemiological cross-sectional study. All subjects underwent pediatric tests based on guidelines on preventiwe examination. A detailed specialist examination was conducted in 569 children and adolescents aged 10–18 selected in the course of the cross-sectional study. Most commonly diagnosed were disorders and diseases of the circulatory system (44.1%), of the osteoarticular system (38.5%), and endocrinological issues (8.8%). The data on individuals affected with cardiovascular problems were analysed in detail. In 38.2% of children and adolescents checked for cardiac murmurs and suspected heart defects, heart defects were excluded and murmurs diagnosed as functional. 21.5% of cardiologically examined subjects were diagnosed with heart defects, 19.1% with hypertension, 16.7% with cardiac arrhythmia, and 4.4% with isolated left ventricular hypertrophy. All were given individual guidance regarding treatment and further specialist health care, and first contact physicians were provided with information on the health status of the child and adolescent population and their needs in the area of preventive medicine and treatment.

**Key words:** Persistent health problems, cardiovascular diseases, children, adolescents

## Introduction

Knowledge on the actual health status of children and adolescents and the conditions behind it forms the groundwork for setting priorities and undertaking structured actions in the area of preventive medicine and health promotion in this population [Braczkowska et al. 2008].

Early recognition of developmental defects, diseases and disorders is a key to effective child and adolescent health care. It enables one to take early preventive actions and apply adequate treatment.

According to the current medical care standards, as set out by the Minister of Health regulation of 28 August 2009, school children are provided with individual health care at their place of residence by basic care doctors and district nurses [http://lex.pl/serwis/du/2009/1133.htm]. Only a few schools have their own preventive health and first aid rooms with a nurse, hygienist or midwife providing students with permanent health care. In most cases, diagnostics and treatment are carried out only when a health problem is reported by a child or noticed by his or her parents and reported to a basic care doctor. As yet, no obligatory standards of preventive actions have been put in place for doctors to follow. This lowers the chances for early detection of health hazards across the population of children and adolescents [Krawczyński 2005].

Studies conducted under the National Health Programme have shown an unsatisfactory level of the health status and health care of Polish children and adolescents [http://www.bpz.gov.pl/dokumenty/proj_npz_2006_15_11102005.pdf]. Accidents and injuries have been found to be the principal life threats, while the use of addictive substances (tobacco, alcohol, drugs) and violence at school have been recognised as the main health hazards. Limited physical activity, poor eating habits and social maladjustment are in turn counted among the most commonly observed inappropriate health-related behaviours [Stańczyk et al. http://www.pfp.edu.pl/index.php?id=wytdzieci].

Circulatory system diseases represent a particular type of health issue, classified in the category of lifestyle diseases. They account for nearly half of all deaths and a large proportion of disabilities in Poland [http://www.mz.gov.pl/wwwfiles/ma_struktura/docs/program_polkard_09062010.pdf]. Prevention and treatment constitute a long-term process that needs to be initiated at a very young age. While cardiovascular mortality is steadily falling, the incidence of these diseases is rising with the increasing life expectancy and ageing of the population. Another contributing factor is the larger population of children and adolescents who were treated with corrective surgery of congenital heart defects, including those that were not operable some years ago.

Among the most common cardiovascular problems reported by children and adolescents to their first contact doctors are chest pains, palpitations and fainting fits. Significant clinical problems that child cardiologists deal with include heart defects, arrhythmias, syncope and high blood pressure [Szydłowski 2003]. Cardiologist consultations are also given to children and adolescents practising sports and those with a family record of sudden cardiac deaths.

## Heart murmur

One of the most frequent reasons for a child to be referred to a cardiologist is heart murmur found during chest auscultation. This symptom, however, cannot be re-

garded as tantamount to a heart defect, as some heart murmurs, called innocent (functional) murmurs, can be found in healthy children. Their occurrence and audibility depend on the stroke volume, width of the large vessel outlets, location of the heart and its distance from the chest. In most children with heart murmur, a distinction between organic and innocent murmur can be made by: thorough interview; proper physical examination using simple tests, such as body position change; performing some physical activities; or auscultation during inspiration and expiration. Additional echocardiography is necessary in the event of uncertainty.

## Angina and sudden cardiac death

Chest pains, often referred to as angina, are among the most frequent reasons for children and adolescents to visit paediatricians and paediatric cardiologists. Only in 4–6% of diagnosed children is the pain found to be of cardiac origin [Aleszewicz-Baranowska et al. 2008]. Sudden deaths rarely occur in children, but when they do, they are often due to pathologies of the cardiovascular system. In contrast to adults, in whom most sudden deaths are provoked by ischaemic heart disease, their causes in children show considerable variation and include myocarditis, cardiomyopathy, cardiac arrhythmia and cardiac conduction defect, as well as some types of heart defects, such as aortic stenosis and congenital coronary vessel anomalies, ischaemic heart disease and myocardial infarction. A retrospective analysis of sudden death cases in children showed that nearly half of them were preceded by cardiac/chest pain, fainting fits or pre-syncope sensations, heart palpitations or decreased tolerance to physical effort. The above symptoms occurred in almost 25–50% of young athletes who died suddenly and in as many as 40–50% of children with recognised heart disease [Milov et al. 1990; Liberthson 1996]. It is believed that most sudden cardiac deaths in children and adolescents are related to disorders of heart rhythm and conduction. Severe cardiac arrhythmias may manifest with fainting episodes during physical effort and strong emotions. They are usually preceded by prodromal symptoms which are a secondary effect of a decreased cardiac output.

Children with suspected cardiac origin of chest pains require a detailed cardiac diagnostic evaluation, including medical interview, physical examination and a number of additional tests, including echocardiography (ECHO), electrocardiography (ECG), 24-hour ECG Holter monitoring (Holter-ECG), cardiac stress test, chest X-ray, and in some cases cardiac catheterisation or electrophysiological cardiac examination. Early diagnosis of circulatory system pathology and implementation of proper treatment, often combined with a change of lifestyle, may prevent a child from sudden cardiac death or lengthen his or her life.

## Syncope

Syncope or fainting is a sudden, brief loss of consciousness due to acute interruption of blood flow to the brain [Lipiec et al. 2004]. 16–20% of children experience

at least one fainting episode before reaching the age of 18 years. In only 50–60% of cases can the cause of fainting be established. Around 6% of all fainting episodes are caused by heart problems. They occur in children with anatomical or functional defects of the cardiovascular system. Faints experienced by a child with previously diagnosed circulatory system pathology may indicate exacerbation of haemodynamic disorders.

Children with tight aortic valve stenosis and hypertrophic cardiomyopathy are particularly at risk [Bieganowska 2008]. Faints related to aortic valve stenosis are believed to be induced by a significant decrease in stroke volume during physical effort and/or anoxaemia of the left ventricle due to blood pressure overload. Syncope can be caused by a low heart stroke volume syndrome or ventricular arrhythmia. A similar mechanism occurs with hypertrophic cardiomyopathy with narrowed left ventricular outflow.

The second group of cardiovascular diseases that predispose to syncope are cardiac arrhythmias. They include recurrent supraventricular tachycardia, ventricular arrhythmias resulting from long QT syndrome, as well as total heart block [Lipiec et al. 2004]. Fainting caused by the above conditions commonly occurs during or after eating.

Vasovagal syncopes account for 60–75% of all fainting fits in children. They happen when the parasympathetic nervous system is stimulated and inhibited at the same time. Stimulation of the vagus nerve causes the heart to slow down its activity, while inhibition of the parasympathetic nervous system leads to relaxation of vessels and reduction of the right ventricular stroke volume, causing blood flow to slow down and a backlog to develop in the peripheral venous system. This results in a decrease in venous return to the heart, right ventricular stroke volume, and lung flow and consequent reduction of the left ventricular stroke volume. Consequently, blood pressure is lowered. The fall in blood pressure is evoked by a vasodepressor response, further deepened by slow cardiac activity, and sometimes leads to sinus arrest. The final outcome of these disorders is a major reduction in the blood flow, acute brain ischaemia and, consequently, a loss of consciousness. Other contributing factors are low blood pressure, fatigue, past infections, ischaemia, malnutrition, and tall height combined with slim body build. Vasovagal syncopes tend to cease on their own as the child grows [Bieganowska 1998; Lipiec et al. 2004].

Approximately 20% of all fainting fits are orthostatic. They occur in children who suddenly change their body position from supine to standing, and are more common in slim, tall individuals, particularly in the period of adolescence. This condition manifests as a feeling of weakness and dizziness or a short-term loss of consciousness combined with facial pallor. The mechanism behind orthostatic fainting involves an immediate drop of blood pressure in the upper part of the body while changing one's body position from supine to standing, leading to acute brain ischaemia and loss of consciousness. A self-induced orthostatic hypotonia may be recognised by diastolic blood pressure lowering upon the change of body position from supine to standing by 30 mmHg and diastolic blood pressure by at least 10 mmHg [Bieganowska 1998; Lipiec et al. 2004].

## Congenital heart defects

The incidence of congenital heart defects is estimated at 5.5–10 per 100 living births. The suspicion of a heart defect in children and adolescents is in most cases based on a heart murmur found during routine paediatric examination. Another group of children referred to cardiologists comprises those with unclear symptoms, such as peripheral cyanosis, chest pain or stinging sensations, shortness of breath, frequent respiratory infections or general developmental disorders. Depending on the haemodynamic situation, a cardiologist sets a plan of diagnosis and treatment, including non-invasive or invasive diagnostic procedures, cardiac observation, or cardiac intervention or surgery. A separate group is composed of children and adolescents with previously diagnosed heart defects, including those before and after surgical treatment and those who have not as yet required treatment. Post-surgery patients require cardiac follow-up due to residual lesions, i.e. anatomical and haemodynamic abnormalities that are constituents of the defect, which are impossible to remove; treatment repercussions, i.e. anatomical and haemodynamic consequences of an applied surgical technique; as well as defect-specific local and systemic complications [Kubicka et al. 2008]. Survival prognosis for post-surgery patients with simple heart defects is the same as for the population-based prognosis. Life expectancy and survival rate are much shorter for all sorts of ventricular dysfunctions, single ventricle, cyanotic heart defect and high blood pressure. Post-surgery or post-intervention heart defect children and adolescents who are candidates for re-operation or re-intervention should be put under particular surveillance. Re-operation involves a higher risk of death and complications than the primary operation, particularly in cyanotic patients. Another growing issue in this group of patients is heart rhythm abnormalities (cardiac arrhythmia) resulting from ventricular enlargements, progressive fibrosis, surgical scar or unsatisfactory post-operative haemodynamics. Post-surgery heart defect patients are also exposed to the risk of sudden cardiac death depending, among other factors, on the type of defect [Kubicka et al. 2008]. The highest risk of sudden death in the late post-operative period occurs in patients with aortic coarctation (CoA), Ebstein's anomaly, corrected transposition of great arteries, aortic valve pathology and tetralogy of Fallot (ToF). Post-operative cardiac arrhythmias constitute another risk factor. Not to be overlooked either is the impact of the physiological ageing process on the circulatory system of post-surgery heart defect patients.

## Cardiac arrhythmia

Cardiac arrhythmia is one of the most frequent reasons for children and adolescents to be referred to a cardiologist. It is also one of the most frequent causes for them to be hospitalised at child cardiology departments [Bobkowski et al. 2010]. The most frequently occurring in children and adolescents is sinus arrhythmia related to the impact of the nervous system and a physiological acceleration of sinus node activity during inspiration and a slow-down during expiration. It is particu-

larly pronounced in adolescents, usually subsiding during physical effort. If no other alarming cardiovascular symptoms are involved, children with sinus cardiac arrhythmia do not require cardiologist consultation.

Such consultation is necessary however with ventricular and supraventricular heart rate variations and heart blocks. The intensity of arrhythmia then has to be evaluated and pathological heart deformation excluded. In some patients, heart rhythm abnormalities may be the first observed symptom of a heart defect or toxic effect of medicines [Bobkowski et al. 2010].

## High blood pressure

High blood pressure (HBP) in the child and adolescent population is a significant paediatric issue. Its prevalence in the under 18 population is estimated at 1–9.8% [Dębiec et al. 1993, Siwik 2007, Widecka 2004; Wojnarowska et al. 2002]. Differences in frequency are due to the diverse environmental and social backgrounds and, in particular, different growth rates, including height and weight, within the studied population [Krzyżaniak 2006].

HBP and obesity are ever more frequent risk factors of atherosclerosis in children. A close correlation has been observed between such factors as weight at birth, body weight growth, systolic blood pressure, lipid disorders and smoking, and the intensity of artherosclerotic lesions in the aorta and coronary arteries [Widecka 2004].

Children and adolescents are found to be increasingly affected by primary hypertension. While data on its prevalence are ambiguous, primary hypertension in this age group is believed to be much more prevalent than thought previously. The probability of primary hypertension being diagnosed is directly proportional to the age of the child. Younger children, in contrast to adults, are predominantly affected by secondary forms of hypertension. However, those over six are sometimes diagnosed with primary hypertension and those over twelve show this form more often than they do the others [Krzyżaniak 2001, Krzyżaniak et al. 2006, Widecka K. 2004]. Around 50% of children with primary hypertension are found to have a family history of the condition.

## Athletic heart

Sport practice makes the circulatory system adapt to intensive physical effort. The resulting deformations depend on the age, gender, duration of practice and the type of sport. Regular sport training induces a reduction in the activity of the sympathetic nervous system, enlargement of heart ventricles and cardiac hypertrophy. A decrease in the tension of the sympathetic nervous system, as an expression of systematic adaptation to physical training, is reflected in sinus bradycardia, AV node-generated rhythm or disorders of atrioventricular and intraventricular conduction [Wożakowska-Kapłon et al. 2006]. Deformations typical for the athletic

heart syndrome subside after the practice is finished. However, latent cardiovascular diseases and cardiac hypertrophy resulting from extensive physical activity pose some risk of complications, with sudden cardiac death as the most serious of them. Therefore, children and adolescents need to be cardiologically examined before they commence and while they practise amateur or professional sports.

## Material

In the ADOPOLNOR project, a randomised population of 4,904 children and adolescents aged 10–18, including 2,431 boys and 2,473 girls, students of primary and secondary schools from the Wielkopolska region, were subjected to an epidemiological cross-sectional study. All subject underwent pediatric tests based on guidelines on preventive examination [Jodkowska, Woynarowska 2002]. A detailed specialist examination was conducted in 569 (11.6%) children and adolescents with chronic diseases who had been selected by physicians conducting screening tests. The most children and adolescents were referred to the specialists with the longest waiting lists.

## Methods

All the qualified individuals underwent medical review and physical examination. When a pathology was found, diagnostic procedures were carried out to provide a final diagnosis and decide on further treatment.

Aside from medical review and physical examination, all children with suspected cardiovascular pathology underwent ECG and ECHO. Children with severe heart rhythm abnormalities were subjected to Holter-ECG, while children with high blood pressure underwent 24-hour blood pressure monitoring (Holter-RR). One child with a congenital heart defect involving an ostium secundum atrial septal defect (ASD II) was diagnosed with transoesophageal echocardiography and cardiac catheterisation.

## Results

Based on information obtained from the parents, 1,967 children, including 922 boys and 1,045 girls, had been under permanent specialist medical care (Table 1).

Most of them used the services of allergologists (n=531), ophthalmologists (n=218), psychologists and school counsellors (n=198), psychiatrists (n=164), orthopaedists (n=149), cardiologist (n=134) and endocrinologists (n=111). The majority were treated by a single clinic, with only one diabetic girl taken care of by

specialists of six different disciplines (endocrinology, pulmonology, ophthalmology, orthopaedics, orthodontics and infectious diseases). One boy under neurological observation had also required periodic psychological consultation.

The scope of information gathered made it possible to make the analysis of such health problems as cardiovascular and osteoarticular system defects and diseases, endocrinological diseases and other conditions that call for continuous medical monitoring (neurological disorders, respiratory, gastro-intestinal and urinary diseases).

Extended clinical tests were carried out in 569 (approx.11,6%) children and adolescents out of 4,904 primary and secondary school children aged 10–18 originally examined in the ADOPOLNOR project (Table 2). Girls represented 52.7%,

Table 1. Students under permanent clinical care

| Students under permanent clinical care n=1967 | | | | |
|---|---|---|---|---|
| Permanent clinical care – medical specialization | boys | | girls | |
| | n | % | n | % |
| Allergology | 272 | 11.44 | 259 | 10.64 |
| Orthopaedics | 61 | 2.56 | 88 | 3.61 |
| Ophthalmology | 96 | 4.04 | 122 | 5.01 |
| Orthodontia | 40 | 1.68 | 65 | 2.67 |
| Laryngology | 27 | 1.14 | 35 | 1.44 |
| Pulmonology | 25 | 1.05 | 23 | 0.94 |
| Cardiology | 65 | 2.73 | 69 | 2.83 |
| Nephrology | 19 | 0.8 | 47 | 1.93 |
| Endocrinology | 43 | 1.81 | 68 | 2.79 |
| Psychology and school counsellors | 105 | 4.41 | 85 | 3.49 |
| Gastroenterology | 10 | 0.42 | 16 | 0.66 |
| Dermatology | 18 | 0.76 | 27 | 1.11 |
| Hepatology | 7 | 0.29 | 7 | 0.29 |
| Neurology | 7 | 0.29 | 6 | 0.25 |
| Psychiatrics | 92 | 3.87 | 72 | 2.96 |
| Diabetology | 7 | 0.29 | 9 | 0.37 |
| Haematology | 7 | 0.29 | 5 | 0.21 |
| Surgery | 6 | 0.25 | 11 | 0.45 |
| Rheumatology | 7 | 0.29 | 6 | 0.25 |
| Cardiosurgery | 1 | 0.04 | 1 | 0.04 |
| Sports medicine | 4 | 0.17 | 3 | 0.12 |
| Vascular diseases | 0 | 0 | 1 | 0.04 |
| Parasitology | 1 | 0.04 | 1 | 0.04 |
| Epidemiology | 0 | 0 | 8 | 0.33 |
| Urology | 2 | 0.08 | 11 | 0.45 |
| All | 922 | 46.9 | 1,045 | 53.10 |

Table 2. Characteristics of the studied children and adolescent population in relation to gender and place of residence

| Number of examined children and adolescents n=569 | | | |
|---|---|---|---|
| Sex | Urban children (63.8%) | Rural children (36.2%) | All |
| Girls | 198 | 102 | 300 (52.7%) |
| Boys | 165 | 104 | 269 (47.3%) |
| All | 363 | 206 | 569 |

boys 47.3% of the studied sample. Urban children accounted for 63.8%, rural children for 36.2% of the sample.

Most frequently diagnosed were disorders and diseases of the cardiovascular system (44.1%), of the osteoarticular system (38.5%) and endocrinological issues (8.8%) (Table 3).

In 38.2% of the children and adolescents referred for cardiologist consultation, cardiovascular pathology was excluded and functional murmur diagnosed instead. 21.5% of subjects were diagnosed with heart defects, 19.1% with hypertension, and 16.7% with heart rhythm abnormalities. In 4.4%, isolated left ventricular hypertrophy was found in ECG and/or ECHO (Table 4).

Defects and diseases of the osteoarticular system were predominantly represented by faulty posture (185 or 84.5% of individuals) (Table 5).

Table 3. Type and number of specialist consultations given to studied group

| Specialist consultation | Number of consultations n=569 | Boys n=269 | Girls n=300 |
|---|---|---|---|
| Orthopaedist | 219 | 108 | 111 |
| Cardiologist | 251 | 126 | 125 |
| Endocrinologist | 50 | 13 | 37 |
| Other: | | | |
| Pulmonologist | 10 | 5 | 5 |
| Laryngologist | 34 | 15 | 19 |
| Neurologist | 4 | 1 | 3 |
| Gastroenterologist | 1 | 1 | 0 |

Table 4. Cardiovascular defects and diseases

| Type of disorder | All n=251 | Boys n=126 | Girls n=125 |
|---|---|---|---|
| Innocent heart murmur | 96 | 34 | 62 |
| Congenital heart defect | 54 | 25 | 29 |
| Hypertension | 48 | 37 | 11 |
| Arrhythmia | 42 | 23 | 19 |
| Left ventricular hypertrophy (observation for cardiomyopathy) | 11 | 7 | 4 |

Table 5. Osteoarticular defects and diseases

| Type of disorder | All n=219 | Boys n=108 | Girls n=111 |
|---|---|---|---|
| Faulty posture (curvature of the spine) | 185 | 85 | 100 |
| Flat feet | 10 | 5 | 5 |
| Chest deformities (chicken chest, funnel chest) | 10 | 10 | 0 |
| Leg length discrepancy | 9 | 5 | 4 |
| Knock-knees | 3 | 1 | 2 |
| Avascular necrosis of femoral head | 2 | 2 | 0 |

Endocrinological issues included mainly obesity (n=17), which was observed in girls twice as often as in boys. Fifteen subjects showed simple goitre, 10 showed thyroiditis, 8 presented with growth failure. Girls prevailed in all patient groups (Table 6).

Children and adolescents with cardiovascular problems were subjected to a detailed analysis. Heart defects were found in 54 cases (Table 7); most of them were mitral defects (n=26). Thirteen children and teenagers showed mitral insufficiency (Fig. 1), and 13 showed mitral valve prolapse with valve insufficiency (Fig. 2a, 2b).

Cardiologist referrals of children and adolescents with isolated mitral insufficiency were based on heart murmurs found during routine paediatric examination. Recurrent respiratory infections were found in the interview in 10 children. None of them reported cardiovascular problems or showed any abnormalities in ECG. Echocardiography revealed a minor anterior mitral cleft in 2 individuals; the others had thickened valves but only 6 exhibited a haemodynamically significant valve insufficiency.

All 13 patients with mitral valve prolapse had been referred to cardiologist consultation because of identified heart murmurs. More than half of them (n=8) reported no cardiovascular issues, and 6 were found to have experienced periodic chest stinging sensations, shortness of breath and low effort tolerance, including 2 with reported palpitations. Echocardiography showed insignificant mitral insufficiency in 12 children and significant mitral insufficiency in 1 child. Two subjects were also diagnosed with supraventricular cardiac arrhythmia (isolated ventricular premature contractions) and 1 with irregularities of the repolarisation period in ECG.

Table 6. Endocrinological diseases

| Type of disorder | All n=50 | Boys n=13 | Girls n=37 |
|---|---|---|---|
| Obesity | 17 | 6 | 11 |
| Goitre | 15 | 5 | 10 |
| Thyroiditis | 10 | 0 | 10 |
| Growth failure | 8 | 2 | 6 |

Table 7. Heart defects

| Type of disorder | All n=54 | Boys n=25 | Girls n=29 |
|---|---|---|---|
| Mitral valve defects including: | | | |
| – MI | 13 | 6 | 7 |
| – MVP + MI | 13 | 7 | 6 |
| ASD II + FOA including: | | | |
| – ASDII | 2 | 1 | 1 |
| – FOA | 10 | 3 | 7 |
| Aortic Valle defects including: | | | |
| – AS | 1 | 1 | 0 |
| – AS+AI | 1 | 0 | 1 |
| – BAV + AS + AI | 5 | 3 | 2 |
| Coronary fistula to pulmonary artery | 4 | 2 | 2 |
| – PS | 1 | 1 | 0 |
| – VSD | 1 | 0 | 1 |
| Postoperative heart defects including: | | | |
| – post PDA | 1 | 0 | 1 |
| – post CoA | 1 | 1 | 0 |
| – post ToF | 1 | 0 | 1 |

MI- mitral insufficiency; MVP- mitral valve prolapse; ASDII- atrial septal defect type II; FOA – foramen ovale apertum; AS – aortic stenosis; AS+ AI – aortic stenosis and aortic insufficiency; BAV – bicuspid aortic valve; PS – pulmonary stenosis; VSD – ventricular septal defect; PDA – patent ductus arteriosus; CoA – coarctation of aorta; ToF – tetralogy of Fallot

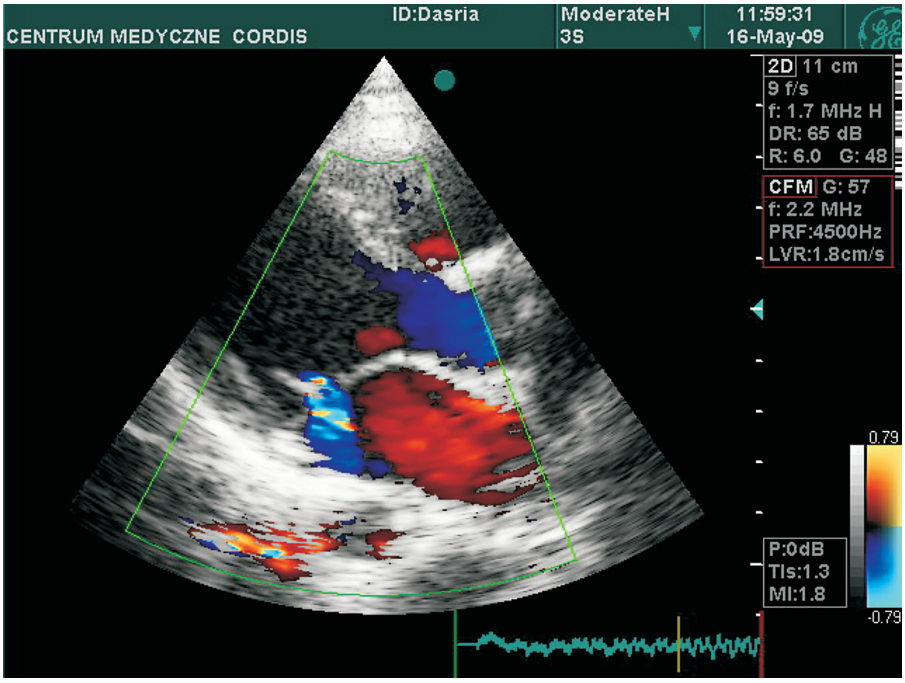



Fig. 1. Echocardiography. Isolated mitral valve insufficiency (MI)

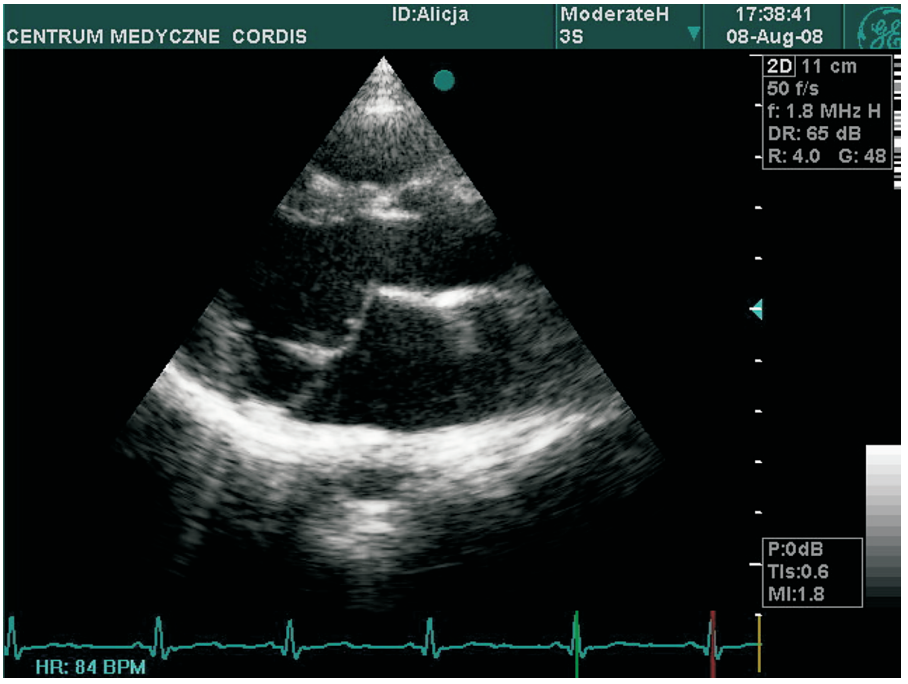



Fig. 2a. Echocardiography. Mitral valve prolapse (MVP)

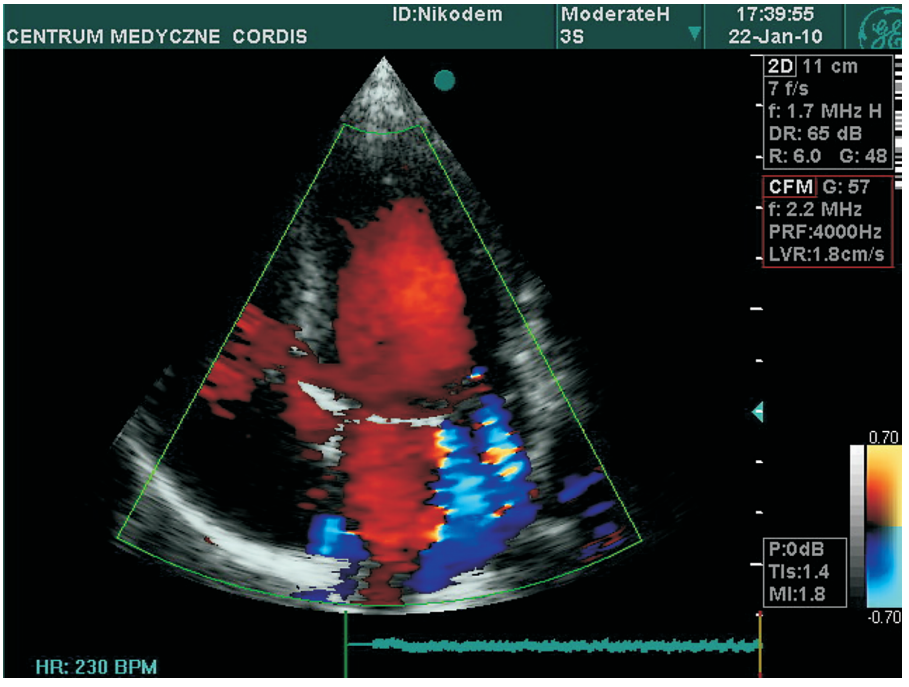



Fig. 2b. Echocardiography. Mitral valve prolapse (MVP) with mitral insufficiency (MI).

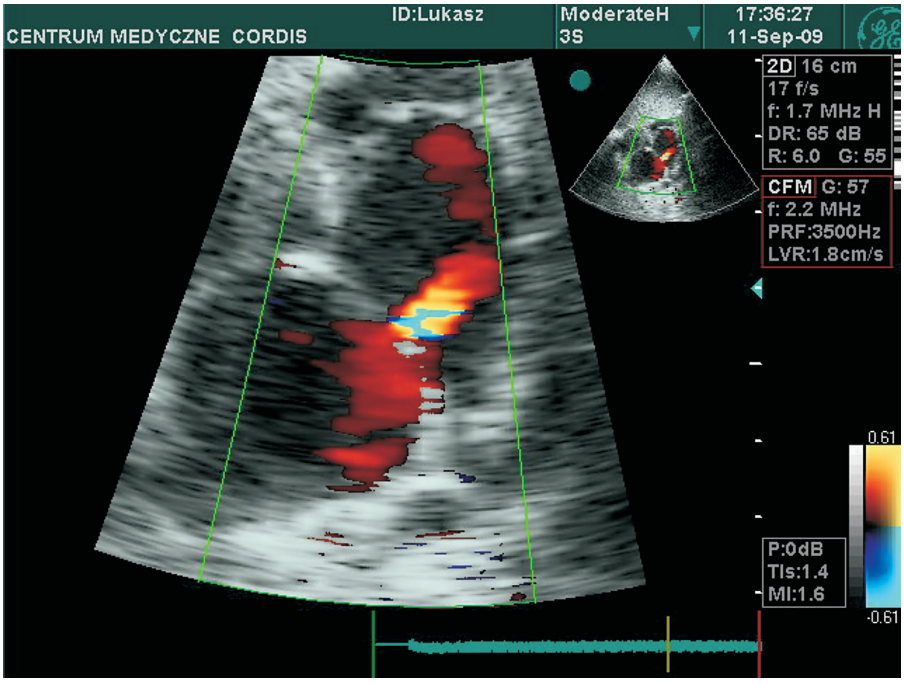



Fig. 3. Echocardiography. Ostium secundum atrial septal defect (ASDII)

Left-to-right ventricular shunt was found in 12 subjects, including across an ASDII in 2 cases (Fig. 3), and through the foramen ovale in the remaining 10. The shunt in a boy with ASD II was haemodynamically significant. Transoesophageal echocardiography was performed in his case (Fig. 4a, 4b) whereby he was qualified for non-operative ASD II occlusion by means of the Amplatzer septal occluder (Fig. 5). The procedure was performed by the Interventional Cardiology Laboratory at the Department of Paediatric Cardiology and Nephrology, Poznan University of Medical Sciences.

Aortic stenosis and aortic valve insufficencywas found in 7 subjects, most of them with bicuspid aortic valve (Fig. 6a–6d).

Four subjects were diagnosed with coronary microfistulas draining to the pulmonary artery (Fig. 7). One child was diagnosed with muscular septal defect (Fig. 8a, 8b).

Only 3 children of the tested group had undergone congenital heart defect surgery, including 1 PDA, 1 CoA, and 1 ToF. The effect of surgery was good in all cases. A 14-year-old girl after PDA surgery showed a normal cardiovascular system. Nor were there any deviations found in ECG and ECHO. A 13-year-old boy after CoA was found to show a systolic gradient in the descending aorta of 14 mmHg, as for now haemodynamically insignificant; heart ventricle sizes were within standard limits, although both ECG and ECHO showed some features of left ventricular hypertrophy. He had no hypertension and his extremity pulse was well palpable. A girl aged 14 after ToF operation showed pulmonary and tricuspid valve incompe-

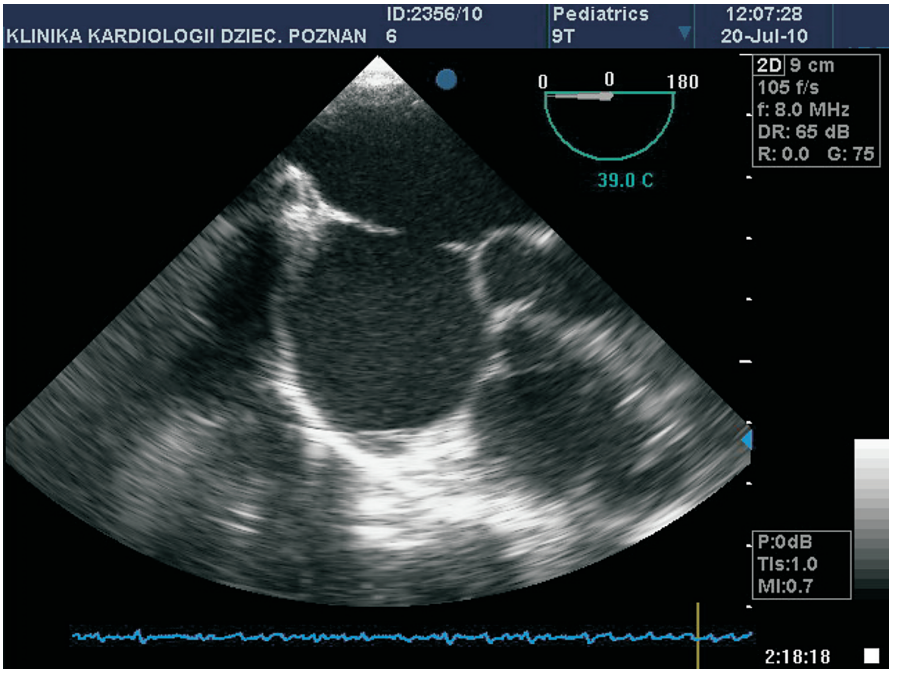



Fig. 4a. Transoesophageal echocardiography. Ostium secundum atrial septal defect (ASDII)

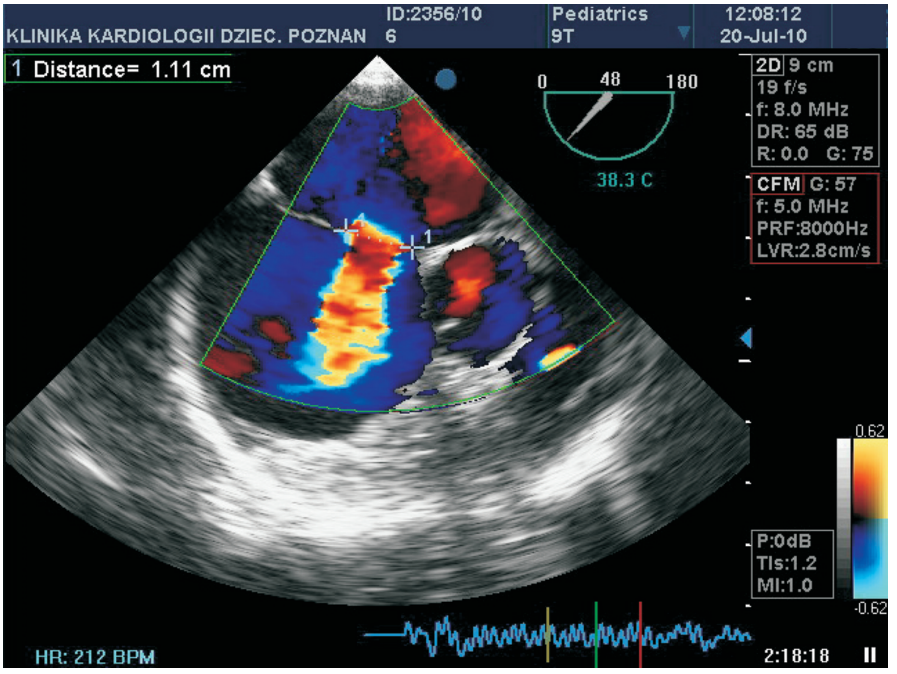



Fig. 4b. Transoesophageal echocardiography. L-R shunt across the ostium secundum atrial septal defect (ASDII)

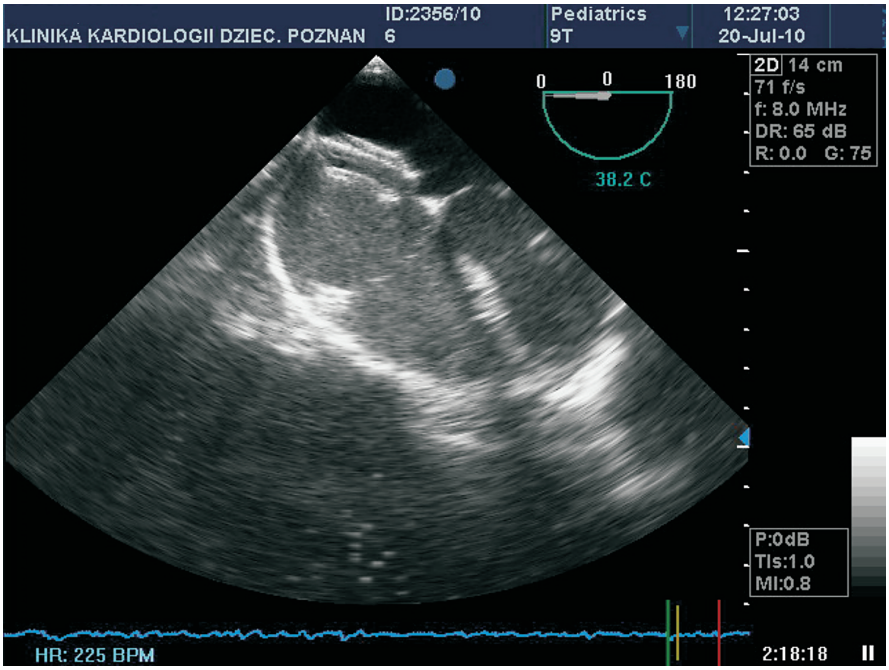



Fig. 5. Echocardiography. Ostium secundum atrial septal defect (ASDII) occluded with Amplatzer septal occluder

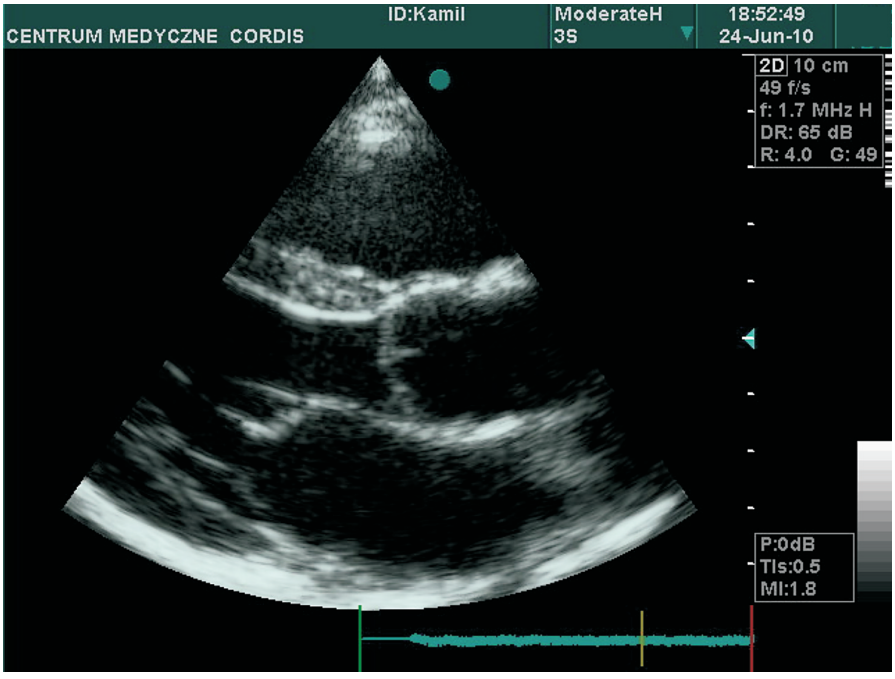



Fig. 6a. Echocardiography. Bicuspid aortic valve

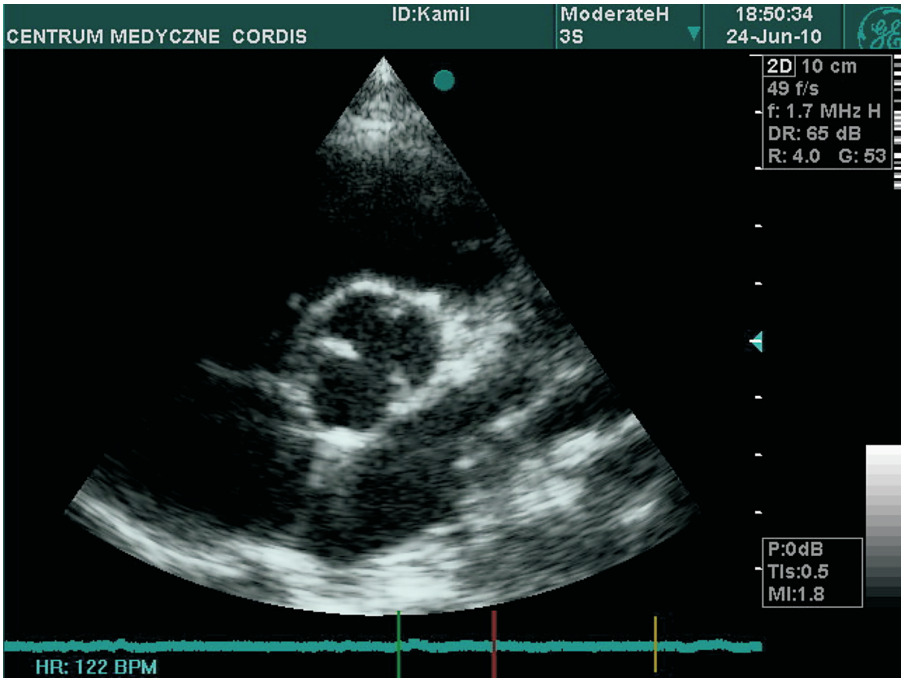



Fig. 6b. Echocardiography. Bicuspid aortic valve

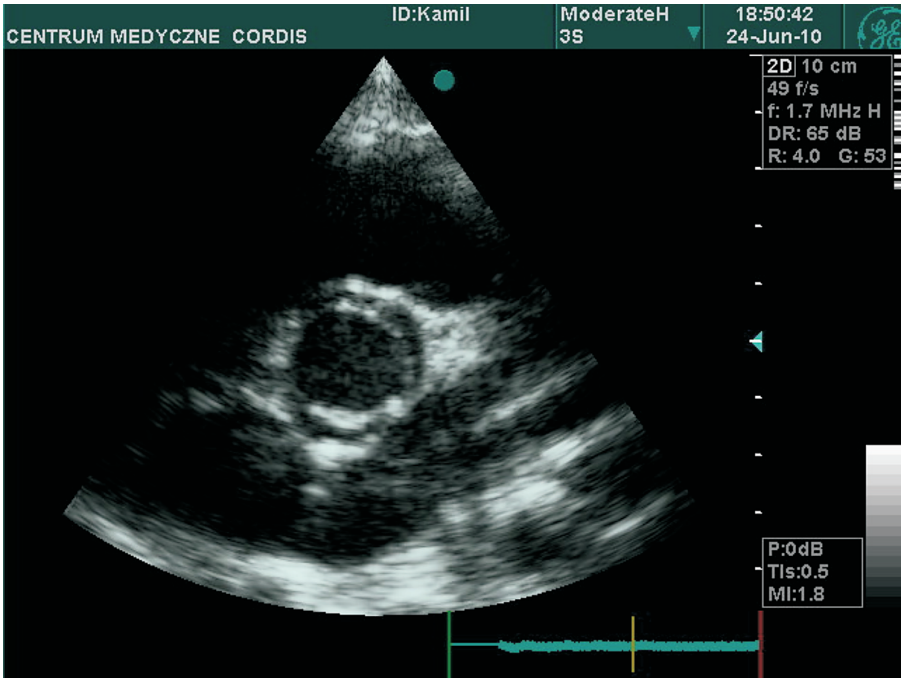



Fig. 6c. Echocardiography. Bicuspid aortic valve (AS)

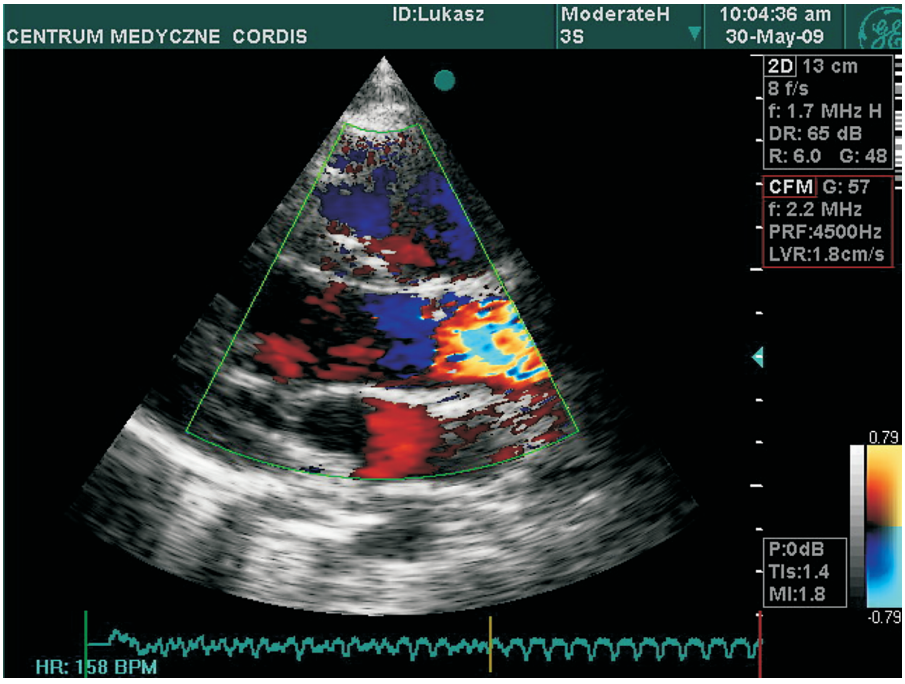
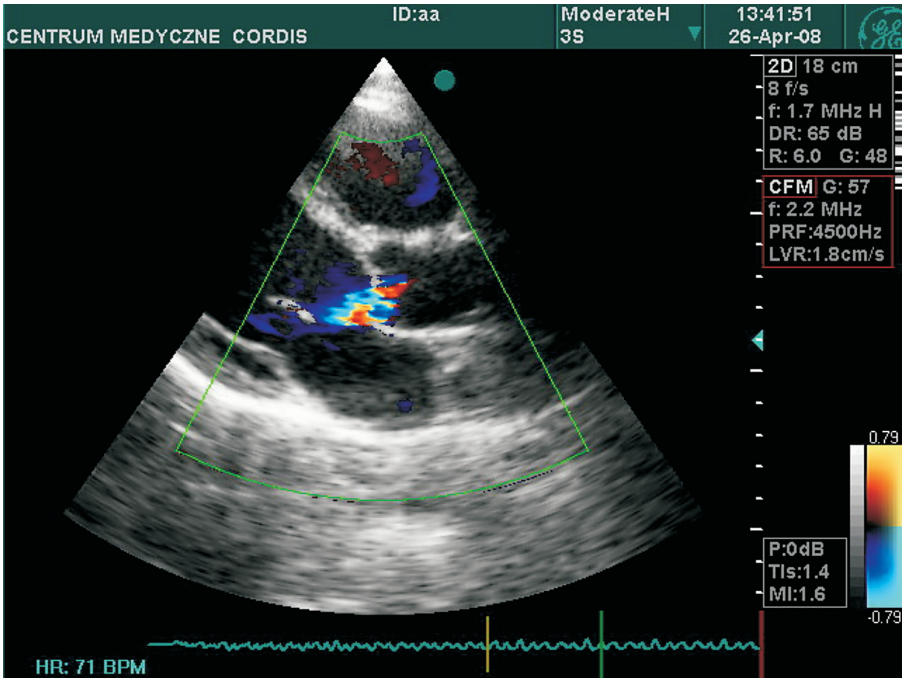



Fig. 6d. Echocardiography. Aortic valve stenosis (AI)




Ryc. 6e. Echocardiography. Aortic valve insufficiency (AI)

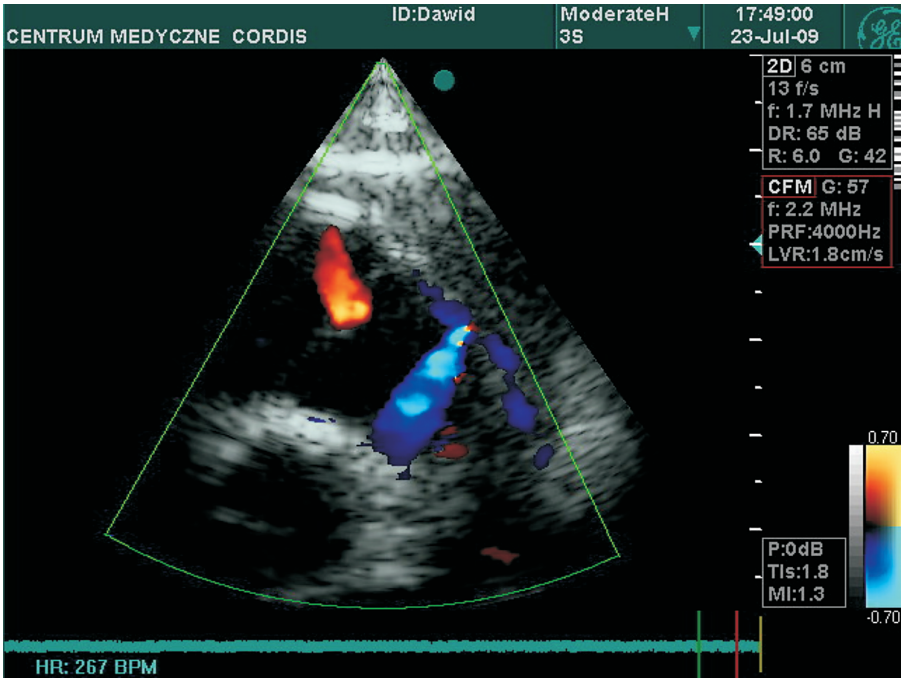


Fig. 7. Echocardiography. Coronary fistula to pulmonary artery

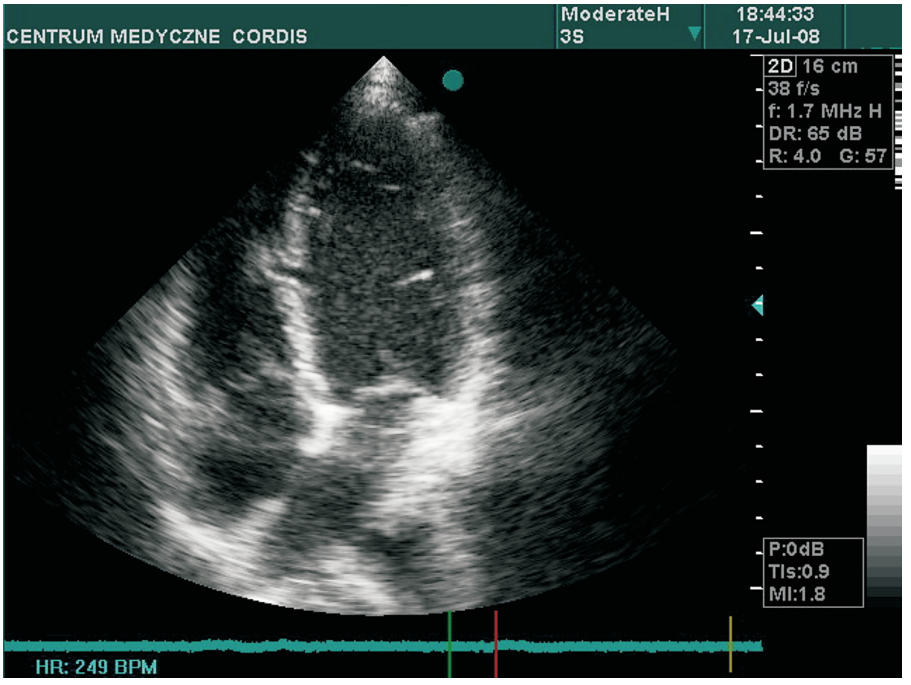


Fig. 8a. Echocardiography. Ventricular septal defect (VSD)

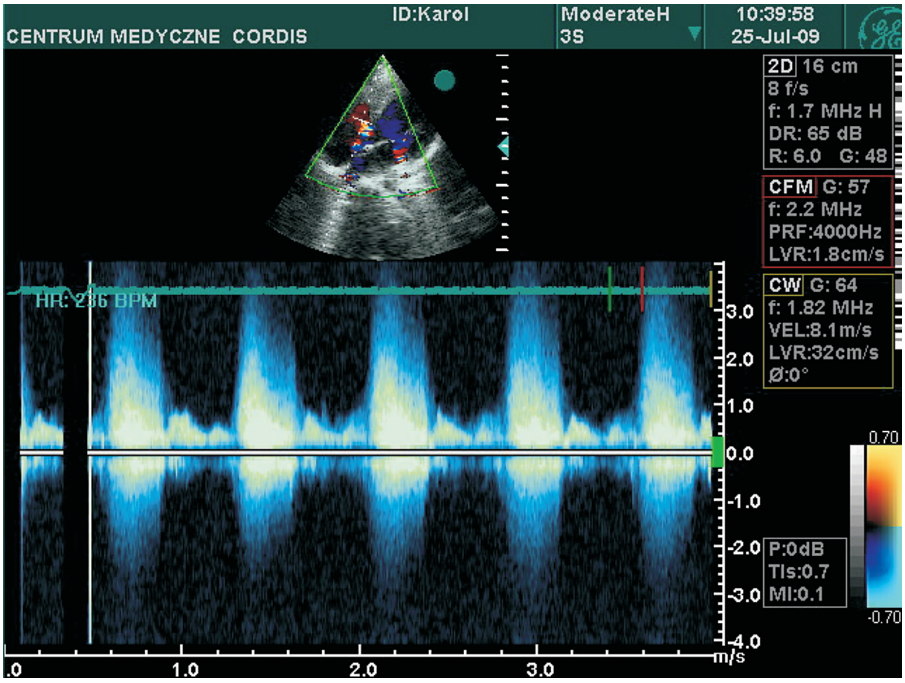


Fig. 8b. Echocardiography. L-R shunt across ventricular septal defect (VSD)

tence, and right ventricular hypertrophy (120% of the average). No significant ventricular-pulmonary gradient was found. The ECG record revealed a total block of the right branch of the bundle of His, duration of QRS complex = 135 ms. No cardiac arrhythmia was identified.

Due to high blood pressure, 48 children and teenagers, including 37 boys and 11 girls, were qualified for extended clinical tests. After complete medical interviews, physical examination, ECG and echocardiography, 30 subjects were recommended to have their blood pressure measurements taken ambulatorily; 18 were referred to a clinic for further diagnosis and treatment.

Thirty-two, including 19 girls and 13 boys, showed cardiac arrhythmias (Table 8). Supraventicular extrasystoles were found in 7 subjects (Fig. 9), ventricular extrasystoles in 5 (Fig. 10). Five were diagnosed with sinus tachycardia, 9 with sinus arrhythmia, and 3 with intraventricular conduction disorders. There were also isolated cases of Lown-Ganong-Levine (LGL) syndrome, Wolf-Parkinson-White (WPW) syndrome and nodal rhythm. Children with heart rhythm abnormalities, apart from ECG and ECHO, were also diagnosed with 24-hour heart activity monitoring by the Holter method (Fig. 11a, 11b).

In 11 subjects, including 7 boys and 4 girls, isolated left ventricular hypertrophy was recognised in ECG and/or ECHO (Fig. 12a, 12b). They were referred to a clinic for further diagnostic examination.

Table 8. Heart rhythm abnormalities

| Type of disorder | All n=32 | Boys n=13 | Girls n=19 |
|---|---|---|---|
| Supraventricular extrasystoles | 7 | 2 | 5 |
| Ventricular extrasystoles | 5 | 2 | 3 |
| Sinus tachycardia | 5 | 2 | 3 |
| Sinus arrhythmia | 9 | 5 | 4 |
| Intraventricular conduction disorders | 3 | 2 | 1 |
| Nodal rhythm | 1 | 0 | 1 |
| LGL syndrome | 1 | 0 | 1 |
| WPW syndrome | 1 | 0 | 1 |

LGL syndrome: Lown-Ganong-Levine syndrome; WPW syndrome: Wolff –Parkinson-White syndrome

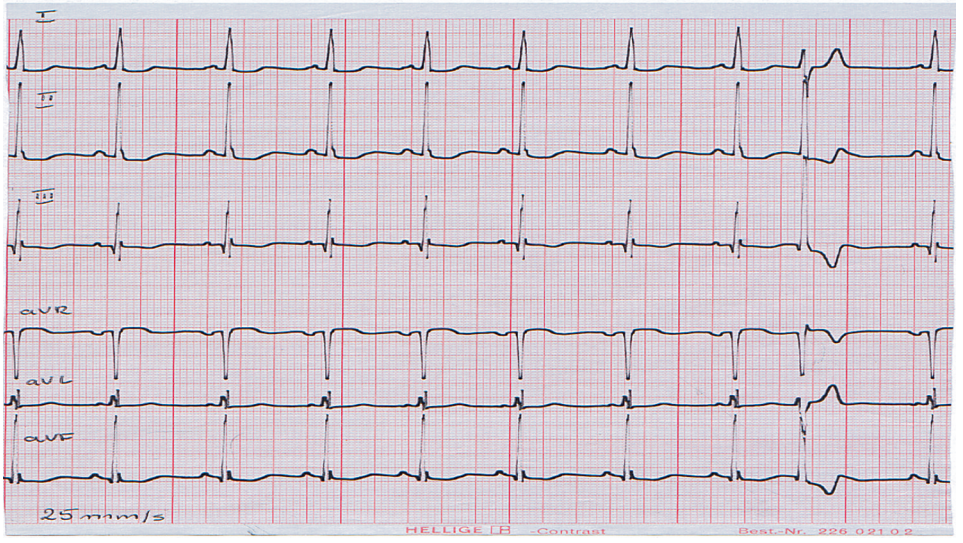



Fig. 9. ECG. Supraventricular cardiac arrhythmia

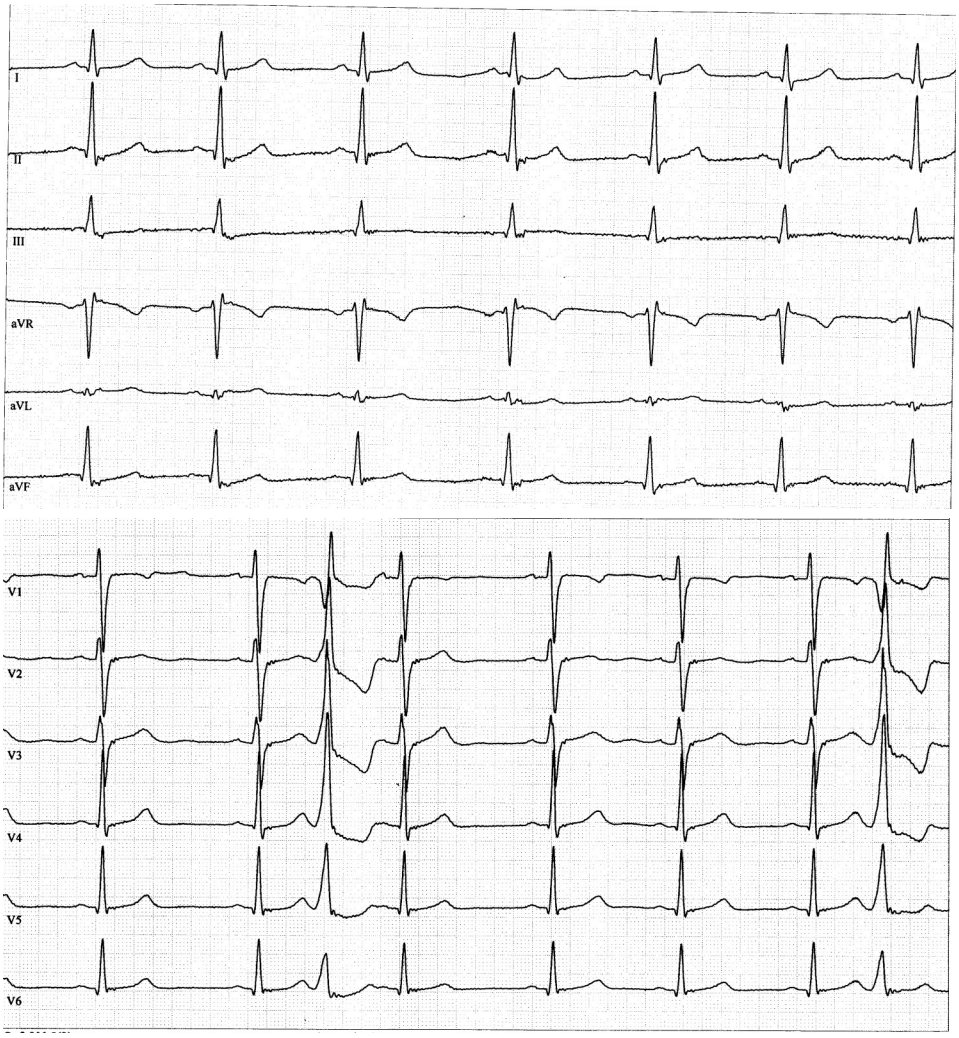



Fig. 10. ECG. Ventricular cardiac arrhythmia

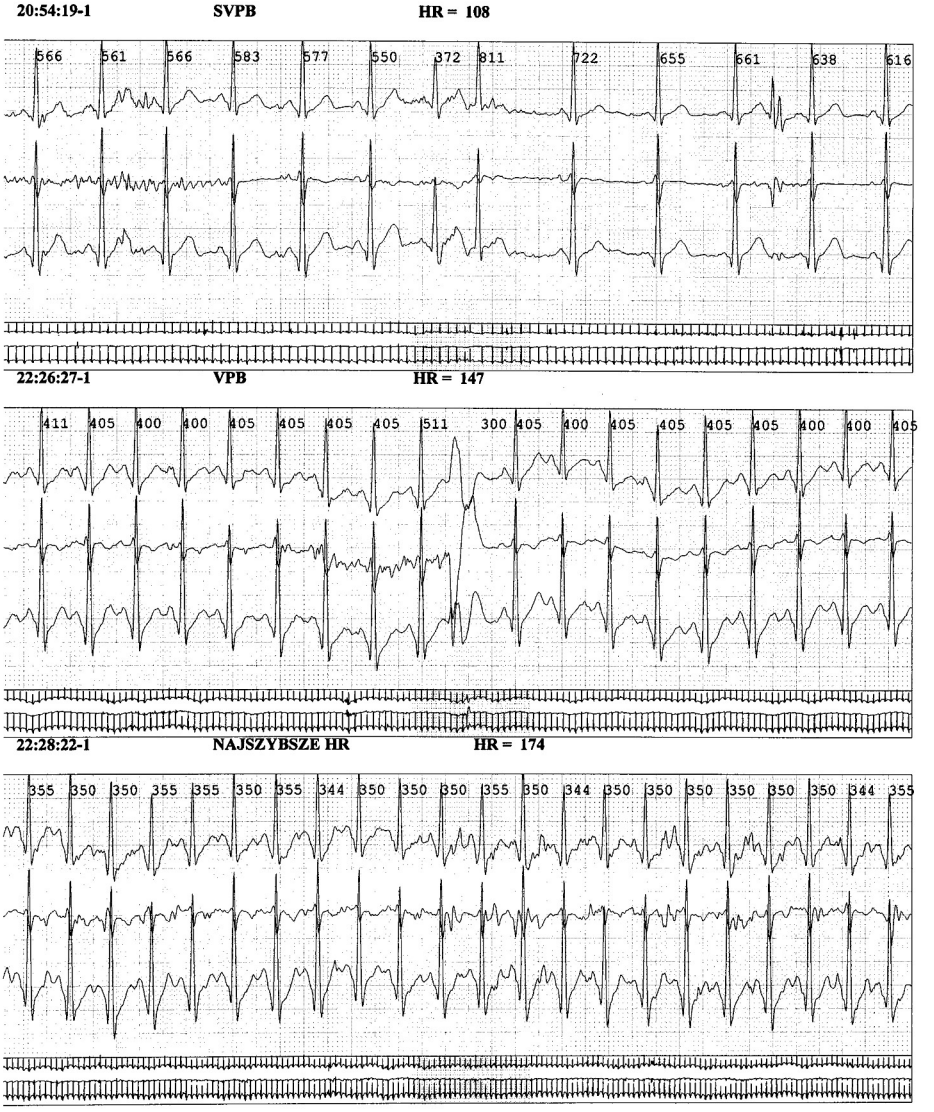



Fig. 11a. 24-hour Holter ECG monitoring

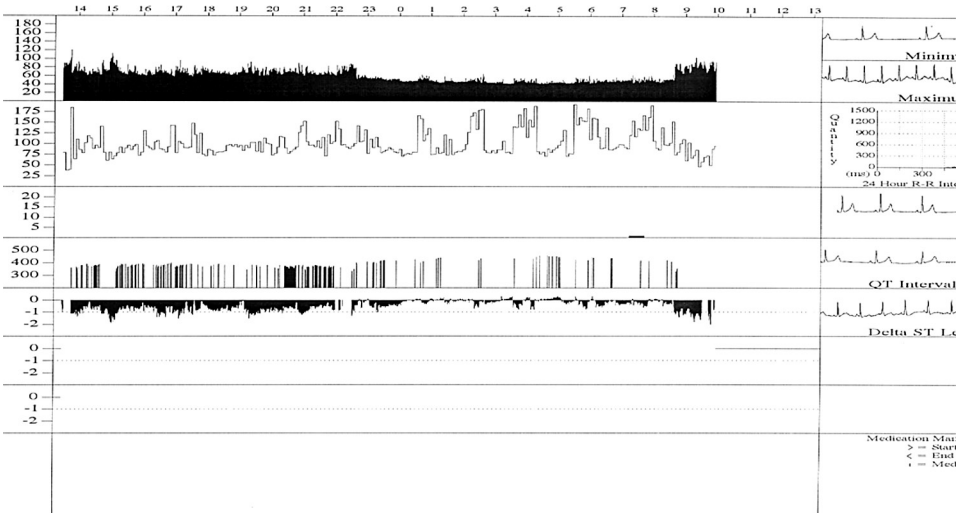



Fig. 11b. 24-hour Holter ECG monitoring

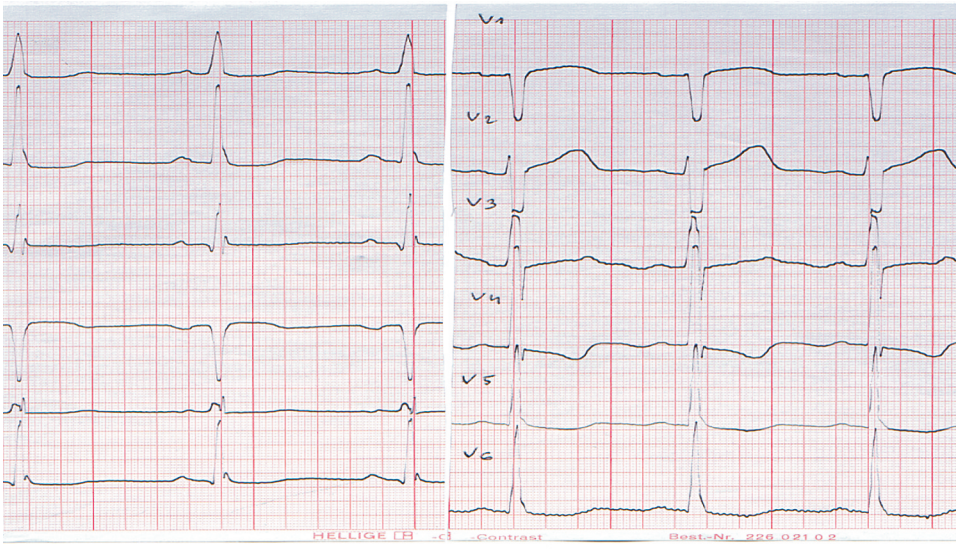



Fig. 12a. ECG. Left ventricular hypertrophy

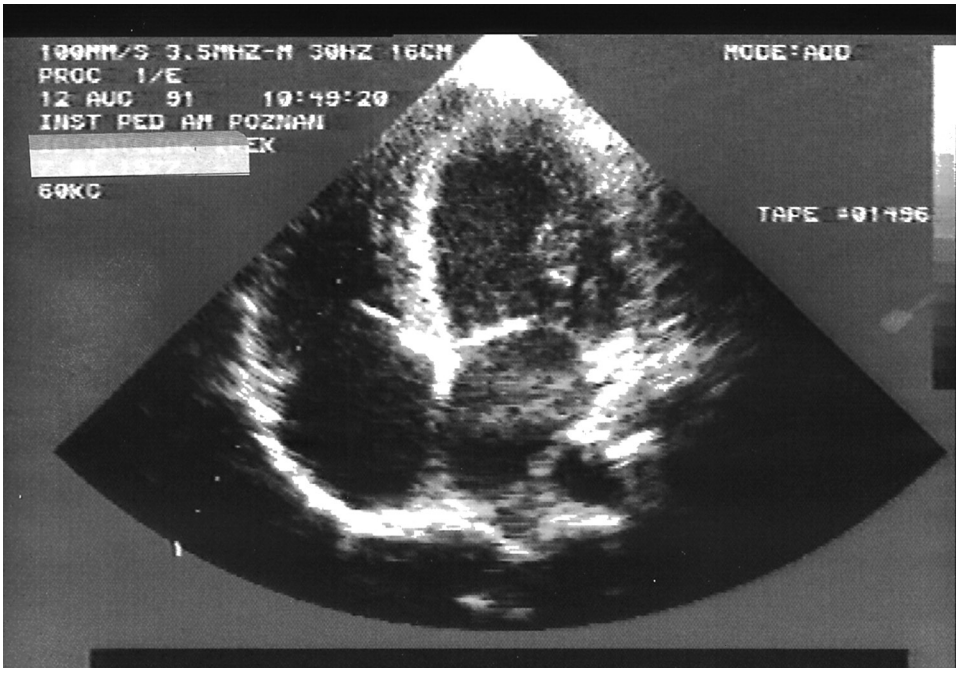



Fig. 12b. Echocardiography. Left ventricular hypertrophy

## Discussion

Heart defects represent a significant paediatric issue. The modern potential for surgical correction of congenital cardiac defects, with age limit lowered to the newborn and infant phase, and an increasing number of surgical interventions for severe defects, previously considered inoperable, poses new challenges for paediatric cardiologists taking care of their patients both before and during surgery. Owing to the rapid development of paediatric cardiology and cardiosurgery, approximately 85% of heart defect children survive into their teenage years, requiring longer period of health care. There are not too many adult patients received by cardiology clinics with congenital heart defects unmodified by earlier surgical or interventional procedures. However, many of those procedures are merely corrective in nature rather than curative, implying defect remnants and repercussions, and sometimes a need for re-operation. In the course of time, clinical problems arise in the form of cardiac arrhythmia, electrical conduction disorders, or haemodynamic complications which may lead to the deterioration of heart performance and a premature death [Trojnarska 2007].

In the group of children and adolescents aged 10–18 studied in the ADOPOLNOR project, heart defects were found in 54 patients. Out of that number, 50 did not require any surgical or interventional treatment, and 1 boy was qualified for occlusion of a septal defect by the Amplatzer occluder. Only 3 children of the tested group had undergone prior congenital heart defect surgery, including 1 PDA, 1 CoA, and 1 ToF. The effects of the operations were good, however, the boy after CoA, and the girls after ToF will require lifetime cardiac follow-up. ToF accounts for an estimated 3.5% of heart defects. Total correction of this complex anomaly has been performed since the mid–1950s. 86% of surgery patients are observed to have a survival rate of more than 30 years. However, due to repercussions of the defect and postoperative complications, patients do not achieve a level of physical performance equal to that of the healthy population. Sudden cardiac deaths are also observed in this group of patients, likely being caused by ventricular cardiac arrhythmias. Risk factors for life-threatening arrhythmia in patients after ToF surgery include a QRS complex of over 180 ms and the occurrence of pulmonary valve insufficiency [Trojnarska et al. 2003]. Another factor that needs to be taken into account for this group of patients is a necessary repair due to pulmonary artery restenosis, significant pulmonary incompetence and progressive right ventricular distension.

In a 14-year-old post-tetralogy girl examined in the ADOPOLNOR project, the QRS complex did not exceed 180 ms, but she was found to show risk factors for cardiac arrhythmia and heart failure, such as pulmonary incompetence, tricuspid insufficiency, and right ventricular hypertrophy. An individual regime of cardiac follow-up examinations was established for her in consultation with her parents.

Aortic coarctation accounts for 5–10% of congenital heart defects. The average length of life for unoperated patients is between 35 and 50 years. The population of those who treated with aortic coarctation surgery, regardless of a good anatomical

and haemodynamic result of the procedure, does not tend to reach an age comparable with that of the healthy population. This is mainly caused by persisting hypertension, ischemic heart disease and heart failure occurring in a large proportion of those patients. As shown by Trojnarska et al. high blood pressure occurs in more than a half of post-operative CoA children and adolescents, being more prevalent in patients who were operated on at older age and not related to the presence of residual gradient in the descending aorta [Trojnarska et al. 2003]. Despite anatomically effective surgery, the deformation of the heart muscle continues to progress constituting an additional life-threatening factor in this group.

A 14-year-old post-operative CoA boy examined in the ADOPOLNOR project showed normal blood pressure and haemodynamically insignificant residual gradient in the descending aorta; however, due to persisting left ventricular hypertrophy he will require constant cardiac observation. Possibly his diagnostic evaluation will have to be supplemented with CT and/or cardiac catheterisation. The boy's parents and first contact doctor were informed about it. Again, as in the case of the girl after the ToF operation, an individual cardiac examination regime was established.

Out of all heart defects identified in the tested children and adolescents, mitral defects represented the largest category. They were found in 26 patients, including isolated mitral insufficiency in 17 and mitral valve prolapse in 13 cases.

The mitral valve prolapse may occur at any age. Although commonly classified as a mild condition, it may lead to life-threatening complications, a fact that needs to be borne in mind whenever mitral valve prolapse is diagnosed [Brot et al. 2000]. Its prevalence is estimated at 4–17% of the whole population, and it is more common in females. Common symptoms exhibited by mitral valve prolapse patients include heart palpitations (66%), long lasting fatigue and low effort tolerance (56%), chest pains (50%), shortness of breath (30%), dizziness (30%), loss of consciousness (23%), and symptoms of anxiety neurosis (12%) [Piwowarska et al. 1997]. Given the diversity of symptoms, it seems useful to make a clinical division of prolapse patients into those affected with palpitations, pain anxiety and fainting.

Out of the 13 patients with mitral valve prolapse, only 1 was diagnosed with significant mitral insufficiency and 2 with supraventricular cardiac arrhythmia. More than half of them (n=8) did not report cardiovascular problems. Only 6 of them had periodic chest pain, shortness of breath, and lower effort tolerance as compared to their peers, and these conditions had not substantially affected their lifestyles.

The diversity of symptoms, often atypical, implies that mitral valve prolapse patients require sufficient attention and appropriate interpretation of their additional test results. Echocardiography is often a key to understanding the mechanism behind the disease. A correct clinical classification enables an appropriate therapeutic approach. It is important that a child and his or her parents are explained the nature of the abnormality, possible symptoms and management guidelines. The patient's diet should be rich in magnesium and mineral salts. He or she should be properly hydrated, as hypovolaemia may cause the disease symptoms to intensify. Physical activity and fitness are important for children with symptoms of constant fatigue. A personalised physical exercise programme should be developed

for them. Recommended activities include aerobics and fast walking for at least 30 minutes [Scordo 1991]. All children with mitral valve prolapse were recommended to use magnesium supplements and the regular care of a cardiologist.

Mitral insufficiency may be caused by widening of the valve ring or abnormalities of cusps, chordae tendineae or papillary muscles. Congenital isolated mitral insufficiency occurs rarely, representing less than 0.5% of all congenital heart defects. One of its causes is anterior mitral cleft, or, extremely rarely, posterior mitral cleft. Isolated mitral insufficiency is also observed in patients with non-infective endocarditis, rheumatism and systemic lupus erythematosus. The clinical symptoms depend on the extent and duration of the insufficiency. Mild or moderate insufficiency usually proceeds without any symptoms, while severe insufficiency produces shortness of breath; cardiac asthma; a tendency to fatigue and weakness; palpitations, especially during physical effort; and finally, symptoms of congestive heart failure.

In the group of children and adolescents studied in the ADOPOLNOR project, minor anterior mitral cleft was found in 2 children. In the other 11 patients with mitral insuffciency, cusps were thickened but there were no grounds to diagnose cougenital heart defects. Ten of them had recurrent respiratory infections, but they had shown no symptoms of rheumatic disorders or infective endocarditis. Children with mitral insufficiency will require the constant care of a cardiologist, and non-invasive circulatory system monitoring.

In the case of congenital aortic stenosis (AS), it is difficult to set an optimal time for surgery as the disease progresses for many years without symptoms or clear indicators for safe observation. AS accounts for 3–6% of heart defects, its incidence being 4 times higher in boys than in girls. In 30% of AS patients, the narrowing is caused by the bicuspid aortic valve,but only 1 per 50 children born with this defect develops a major aortic stenosis or insufficiency. In patients with a normal three-leaflet valve, the narrowing is caused by partial consolidation of the leaflets. The narrowing may be a separate condition or combined with insufficiency.

Congenital AS may progress in two ways: with early symptoms, in children born with a critical narrowing, or without any symptoms, even up to the fourth decade of life [Siwińska 1999]. In the case of severe narrowing, the muscle of the left ventricle is already damaged in the prenatal or infancy period and remains so thereafter. In most children, however, the defect causes no symptoms, even with a large degree of narrowing; hence they live a normal life throughout their childhood and adolescence.

In contrast to adults with acquired AS children do not often report chest pain, shortness of breath or low effort tolerance. Nor do they often show symptoms of heart failure. The first clinical manifestation of the defect may be a sudden cardiac death without any warning signs. This occurs in approximately 10–15% of patients. There is no effective pharmacological treatment of AS. Invasive treatment involves such methods as balloon valvuloplasty, surgical valvulotomy and valve replacement.

The clinical status of AS patients depends on the intensity, growth rate and nature of the defect. In children with congenital stenosis, the disease may cause no

symptoms for a long time. Some children, like AS adults, report chest pain, dizziness, palpitations and coronary pain.

For patients with a complex aortic defect, the clinical status depends on the extent of stenosis and insufficiency and the growth rate of haemodynamic disorder progression [Siwińska 1999]. Reported ailments include pain, dizziness and palpitations. Upon the occurrence of left ventricle disorders, patients start to report fatigue, shortness of breath on exertion, and cardiac asthma. Coronary pain may occur in patients with a complex aortic defect with no anatomical lesions in coronary vessels.

In the group of children and adolescents examined in the ADOPOLNOR project, aortic valve stenosis was diagnosed in 7 patients, including 1 narrowing of a three-leaflet aortic valve, 1 stenosis and insufficiency of a three-leaflet valve, and 5 bicuspid aortic valves without stenosis or insufficiency. Aortic stenosis had not been suspected in any of the children. They had been referred for cardiology consultation on account of a functional cardiac murmur in order to exclude a heart defect.

Many of the recommendations for adults with acquired AS may very well be applied to younger patients, but there some important differences that need to be underlined. In the childhood period, the valve ring and cusps have to grow along with the whole body. The narrowing may occur when the growth rate is delayed. The progression rate during childhood and adolescence may be different than in adults with an acquired defect [Bonow et al. 2006]. Many patients with a bicuspid aortic valve exhibit disorders of vascular connective tissue, such as the loss of elastic tissue, which may give rise to expansion of the aortic bulb or ascending aorta, even without a haemodynamically significant stenosis or insufficiency of the aortic valve. Such patients are threatened with aortic dissection. Beta-blocker treatment has been reported to effectively inhibit aortic enlargement; the studies, however, were conducted for Marfan syndrome patients rather than those with bicuspid aortic valve. Repair operations of an aortic bulb or replacement of a descending aorta are recommended for patients with a greatly enlarged aortic bulb or descending aorta. A number of factors need to be taken into account on such occasions, including the patient's age, the relative size of the aorta and its bulb, the structure and activity of the aortic valve and the experience of the surgical team [Bonow 2006].

Coronary artery fistulas are defects of the coronary circulation involving anomalous connections between coronary arteries and heart ventricles or large vessels. Most of them are congenital, although they can also be iatrogenic in nature as complications after cardiac surgery, such as aortic valve replacement, angioplasty procedures or post-traumatic. The incidence of coronary artery fistulas is hard to evaluate precisely, as approximately 75% of affected individuals may experience no clinical symptoms of the defect. It is assumed that coronary vessel anomalies occur in 1–15% of the total population and 0.1–0.2% of patients managed with coronarography [Burchardt et al. 2008; Kurzawski et al. 2010; Skalski et al. 2003]. A vast majority, 87%, are the forms involving abnormal origin and course of coronary vessels. The remaining 13% are coronary fistulas, accounting for less than 1% of all congenital heart defects. Every third case or so may be accompanied by another congenital heart defect. As a rule, patients with isolated congenital coronary fistulas do not show any symptoms in their childhood and early adulthood, the fre-

quency of health problems increasing with age [Burchardt et al. 2008; Janion et al. 2004; Manghat et al. 2005; Said et al. 2006]. Initially, heart failure symptoms prevail. Older patients often manifest myocardial infarction, ventricular cardiac arrhythmia, infective endocarditis, pulmonary hypertension and least often sudden cardiac death [Manghat et al. 2005; Qereshi 2006]. Most fistulas enter the right ventricle and coronary sinus. Only 7% are connected with the pulmonary artery. Currently, there are no guidelines on how to proceed with diagnosed fistula patients. Most experts believe that patients with symptomless small fistulas do not require surgical intervention or percutaneous fistula closure.

In the group of children studied in the ADOPOLNOR project, coronary microfistulas (<1mm) draining to the pulmonary artery were found in 4 subjects. Children had reported no ailments. They had been referred to a cardiologist on account of an identified heart murmur to exclude a heart defect. The children will remain under cardiac monitoring.

Cardiac arrhythmias were found in 32 children and adolescents. The group included children with ventricular and supraventricular extrasystoles, sinus brachycardia, sinus arrhythmia, intraventricular conduction disorders, LGL syndrome, WPW syndrome and nodal rhythm. Cardiac arrhythmias in the form of ventricular premature contraction occur in approximately. 15% of children and adolescents, often in those without anatomical heart pathology. Usually, they do not lead to any ailments or heart failure symptoms. They can be caused by hypomagnesaemia or be a first observed symptom of a heart defect or cardiomyopathy [Bobkowski et al. 2010].

All children with supraventricular extrasystoles examined in the ADOPOLNOR project were diagnosed with hypomagnesaemia and for that reason were prescribed magnesium supplements and referred for further cardiac observation.

The most prevalent symptomatic arrhythmia in children is supraventricular tachycardia. It may occur in a latent form or as recurrent episodes. In most children with this type of arrhythmia, no heart pathology was found. However, some heart defects, such as Ebstein's anomaly, corrected transposition of great arteries, mitral valve prolapse, and cardiomyopathy, do predispose to it. Each child with tachycardia must be checked for myocarditis and hyperthyroidism. Out of all examined children and adolescents, 5 reported palpitations in their medical interview, but these were not confirmed by objective examination. During physical examination, they were only found to present with insignificant sinus tachycardia at the beginning of the procedure. Then it disappeared as the examination continued. Tachycardia was not diagnosed by the Holter-ECG either. Nor were any cardiac defects of hyperthyroidism identified.

Each child showing premature ventricular contractions requires consultation with a cardiologist to evaluate the degree of the arrhythmia and exclude possible underlying causes, particularly heart tumours, myocarditis, hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, e.g. long QT syndrome, and possible toxic effects of medicines taken. The cardiac stress test is recommended for children with ventricular arrhythmia in order to evaluate the intensity of arrhythmia and corrected QT time during physical effort [Bobkowski et al. 2010]. Even numerous premature ventricular contractions usually do not require anti-ar-

rhythmia treatment, if they occur, in a child without heart pathologies and do not exhibit any clinical symptoms, including those of heart failure. The only recommendation is to take magnesium supplements. An increase in ventricular arrhythmia severity on exertion is a sign of poor prognosis and usually an indication to start anti-arrhythmia treatment. Similar treatment is implemented in children with ventricular extrasystoles combined with another heart defect. Children with ventricular extrasystoles children are rarely candidates for ablation.

This condition was diagnosed in 5 children. All of them had a normal heart ECHO. Following a Holter-ECG, 2 were given additional beta-adrenolytic treatment and 3 were prescribed to take magnesium preparations. All the children were referred to cardiac care units.

Nine children were diagnosed with sinus arrhythmia. The ECHO was normal in all cases. Their sinus arrhythmia passed after physical effort. Children affected with this type of arrhythmia do not require cardiac treatment.

There was also no need to extend the diagnostic process and implement treatment in 3 children with conduction disorders in the right branch of the bundle of His or 1 with diagnosed nodal rhythm. Recommendations were limited to ECG to be performed once a year by a first contact doctor and periodic cardiac evaluation.

Cardiologist care and pharmacological treatment are however needed for 2 children with periodic palpitations and pre-excitation syndrome – 1 with WPW syndrome and 1 with LGL syndrome. Neither of them was diagnosed with ECG during a palpitation episode. As both syndromes may lead to a life-threatening atrioventricular tachycardia, the children were referred for electrophysiological treatment and ablation.

Blood pressure (BP) levels in children are lower than in adults and rise linearly with age and growth up to the age of puberty. In the periods of growth spurts, blood pressure rises proportionally to the accelerated body growth. Epidemiological studies have enabled the distribution of normal BP levels in various age groups to be established and centile grids necessary to interpret measurements in relation to gender, age and height to be developed [Krzyżaniak et al. 2006; Widecka 2004].

HBP was found in 48 subjects, including most of the boys. Congenital heart defects were excluded in all cases. In 30, ECG and ECHO showed no myocardial hypertrophy. They were instructed to take daily BP measurements in a home setting and appointed for follow-up examination in a cardiac unit. The other children who showed hypertension and left ventricular hypertrophy in ECG and/or ECHO were referred to a clinic for comprehensive diagnosis. Twelve subjects were found to be overweight and 6 obese. All of them had high blood pressure. Fourteen were prescribed non-pharmacological and 4 pharmacological treatment.

Eleven subjects, including 7 boys and 4 girls, aged 18 were diagnosed by ECG and/or ECHO with different degrees of left ventricular hypertrophy. They did not report any major health problems apart from periodic chest stinging sensations. Non-invasive examination excluded heart defects. High blood pressure was not found either. Physical examination revealed muscle hypertrophy. They had not taken anabolic steroids, though. All of them had worked out or practised judo for several years. Sport training causes changes in the morphology and activity of the

heart, known as athletic heart. They include an increase in the heart weight and size and the changes in the regulation of the parasympathetic and sympathetic nervous system. This leads to an increase in the blood volume ejected from the heart ventricles at a single stroke. The intensity and nature of heart remodelling depend on the kind of activity practised, the athlete's heart, and the duration and intensity of training [Wożakowska-Kapłon et al. 2006]. The degree of heart adaptation to endurance training is individually variable. Therefore, caution should be taken in defining athletic heart changes as typical, physiological, reversible or safe for lie. This requires appropriate cardiological experience. There is no evidence confirming a negative impact of intensive physical exercise on the circulatory system of a person with a normal heart. Sudden deaths occasionally occurring among athletes are are often caused by undiagnosed heart defects.

It cannot be excluded that the cardiac hypertrophy found in 11 young people examined in the ASDOPOLNOR project was linked to their sport activity. Their circulatory systems, however, will need to be monitored by sport doctors and paediatric cardiologists.

## Conclusion

1. The greatest health care needs in the Wielkopolska region apply to children and adolescents with allergic, ophthalmological, psychological, psychiatric, orthopaedic, cardiac and endocrinological problems.
2. It is necessary to improve the availability of orthopaedic, endocrinological and cardiac health care services in the studied region.
3. Among the most frequently reported chronic cardiovascular problems in children and adolescents are heart defects, cardiac arrhythmias and high blood pressure.
4. Children and teenagers practising sports should have their circulatory systems monitored for possible changes referred to as athletic heart, which need to be properly identified, and conditions that might lead to a sudden cardiac death.

## References

Alaszewicz-Baranowska J., Potaż P., Komorowska-Szczepańska W.: Bóle serca i nagłe zgony kardiologiczne u dzieci. *Choroby Serca i Naczyń* 2006; 3: 160–166.

Bieganowska K.: Najczęstsze przyczyny i diagnostyka omdleń u dzieci i młodzieży. *Standardy Medyczne* 2008; 10, supl. 32: 61–65.

Bobkowski W., Baszko A., Stefaniak M., Siwińska A.: Aktualne zasady postępowania w zaburzeniach rytmu serca u dzieci. *Lekarz* 2010; 9: 12–20.

Bonow R.O., Carabello B.A., Chatterjee K. et al.: Postępowanie w zastawkowych wadach serca – wady zastawki aorty. Aktualne (2006) wytyczne American College of Cardiology i American Heart Association opracowane na podstawie: ACC/AHA 2006 guidelines for the management of patients with valvular heart disease. Available at: http://www.

mp.pl/artykuly/index.php?aid=30365&_tc=0A94B4FA516C48418545E145427E2835&print=1 (14.06.2011). A Report of the American College of Cardiology/American Heart Association Task Force on practice guidelines (Writing Committee to Revise the 1998 Guidelines for the Management of Patients With Valvular Heart Disease) developed in collaboration with the Society of Cardiovascular Anesthesiologists. JACC 2006; 48: e1-e148.

Braczkowska B., Cyran W., Braczkowski R., Kowalska M.: Problemy zdrowotne młodzieży szesnastoletniej – uczniów szkół ogólnokształcących w Oświęcimiu. *Prob Hig Epidemiol* 2008; 89: 359–366.

Brot T.S., Bissinger A.P.: Objawowe i bezobjawowe wypadanie płatka zastawki dwudzielnej. *Forum Kardiologów* 2000; 3: 82–87.

Burchardt P., Angerer D., Wysocki H.: Wrodzone przetoki naczyń wieńcowych. *Kardiol Pol* 2008; 66: 992–994.

Dębiec B., Godzisz M., Pokuszyńska K. et al.: Zachowanie się wartości ciśnienia tętniczego krwi młodzieży kończącej szkolę podstawową w Łodzi i regionie łódzkim, cz. I. *Zdr.Publ* 1993; 106: 14–17.

Janion M., Wesołowska K., Ciuraszkiewicz K. et al: Anomalia tętnic wieńcowych pod postacią przetok wieńcowo-płucnych – częstość występowania, patofizjologia i znaczenie kliniczne. *Folia Kardiol.* 2004; 11: 383–387.

Krawczyński M.: Aktualne problemy zdrowotne i organizacyjne opieki nad dziećmi i młodzieżą. *Pediatr Pol* 2005; 80: 947–953.

Kubicka K., Kawalec W. (Eds.): *Wady wrodzone serca u młodocianych i dorosłych.* Poradnik dla lekarzy podstawowej opieki zdrowotnej. Wydawnictwo Lekarskie PZWL, Warszawa 2008.

Kurzawski J., Domagała S., Peciak J. et al.: Przetoka wieńcowo-płucna z ostrym zespołem wieńcowym z uniesieniem odcinka ST – problem kliniczny i diagnostyczny. *Kardiol Pol* 2010; 68:446–449.

Krzyżaniak A.: Współczesne problemy zdrowotne dzieci i młodzieży. *Prob. Hig* 2001; 73: 38–43.

Krzyżaniak A., Kaczmarek M., Palczewska I. et al.: Propozycje norm ciśnienia tętniczego u dzieci i młodzieży. *Pediatr Pol* 2006; 2: 107–1111.

Lipiec A., Konarska-Matysiak D.: *Omdlenia u dzieci i młodzieży – jeden objaw wiele przyczyn*. Available at: http://www.alergia.org.pl/lekarze/archiwum/04_01/pdf/2004_ 0107.pdf (14.06.2011).

Liberthson R.R.: Sudden Heath from cardiac causes In children and young adults. *NEJM* 1996; 334: 1039–1044

Manghat N.E., Morgan-Hughes G.J., Marshall A.J. et al.: Multidetector row computed tomography: imaging congenital coronary artery anomalies in adults. *Heart*, 2005; 91: 5115–1522.

Milov D.E.,Kantor R.J.: Chest pain In teenagers. When is it significant? *Postgrad. Med.* 1990; 88:145–154.

*Narodowy Program Wyrównywania Dostępności do Profilaktyki i Leczenia Chorób Układu Sercowo-Naczyniowego na lata 2010–2012 POLKARD*. Available at: http://www.mz. gov.pl/wwwfiles/ma_struktura/docs/program_polkard_09062010.pdf (14.06.2011).

Piwowarska W., Kiliński M.: *Objawowe i bezobjawowe wypadania płatka zastawki dwudzielnej*. Bel. Corp. Warszawa 1997.

Qereshi S.A.: Coronary arteria fistulas. *Orphanet J Rare Dis* 2006; 21: 51.

Rozporządznie Ministra Zdrowia z 28 sierpnia 2009 r. w sprawie zakresu i organizacji profilaktycznej opieki zdrowotnej nad dziećmi i młodzieżą (Dz.U. 2009, nr 139, poz. 1133).

Said S.A., Lam J., van der Werf T.: Solitery coronary artery fistulas: a congenital anomaly In children and adults. A contemporary review. *Congenit Heart Dis* 2006; 1: 63–76.

Scordo K.A.: Effects of aerobic exercise training on symptomatic women with mitral valve prolapse syndrome. *Am. J Cardiol.* 1991; 67: 863–868.

Siwik P.: *Częstość i uwarunkowania nadciśnienia tętniczego dziewcząt w wieku szkolnym w wybranych miastach województwa śląskiego* (praca magisterska). Śląski Uniwersytet Medyczny, Katowice 2007.

Skalski J.H., Kovalenko I.: Przetoki naczyniowe. In: Skalski J., Religa Z. (eds), *Kardiochirurgia dziecięca.* Wydawnictwo Naukowe Śląsk, Katowice 2003: 381–392.

Siwińska A.M.: *Echokardiograficzne wskaźniki czynności skurczowej i rozkurczowej serca i ich znaczenie rokownicze u niemowląt, dzieci i młodzieży z wrodzonymi wadami zastawki aortalnej oraz zwężeniem cieśni aorty*. Praca habilitacyjne. UM Poznań 1999.

Stańczyk J., Kierkowska B., Podolec P., et al.: *Wytyczne Grupy Roboczej Polskiego Forum Profilaktyki Chorób Układu Krążenia dotyczące profilaktyki chorób sercowo-naczyniowych u dzieci i młodzieży*. Available at: http://www.pfp.edu.pl/index.php?id=wytdzieci (14.06.2011).

Szydłowski L. (ed.): *Problemy kardiologiczne u nastolatków*. II Konferencja Szkoleniowo-Naukowa Sekcji Kardiologii Dziecięcej Polskiego Towarzystwa Kardiologicznego. Streszczenia. Częstochowa 19–21 września 2003 r.

Trojnarska O., Siwińska A., Wachowiak-Baszyńska H. et al.: Zaburzenia rytmu serca oraz zmienność rytmu zatokowego i dyspersja QT a niedomykalność zastawki pnia płucnego i czas trwania zespołu QRS u dorosłych chorych po całkowitej korekcji tetralogii Fallota. *Folia Cardiologica* 2003; 10: 185–193.

Trojnarska O., Szyszka A., Ochotny R., Siwińska A., Cieśliński A.: Zwężenie cieśni aorty. Ciśnienie tętnicze, masa i funkcja lewej komory po skutecznej operacji. *Kardiol. Pol.* 2003; 59: 317–319.

Trojnarska O.: *Dorośli z wadami wrodzonymi serca – wzrastająca populacja pacjentów. Podstawowe problemy diagnostyczne i terapeutyczne*. Ośrodek Wydawnictw Naukowych. Poznań 2007: 11–28.

Widecka K.: Nadciśnienie tętnicze u dzieci i młodzieży- coraz większy problem medyczny. *Choroby Serca i Naczyń* 2004; 1: 88–91.

Woynarowska B, Jodkowska M.: Test przesiewowy do wykrywania podwyższonego ciśnienia tętniczego. In: Jodkowska M., Wojnarowska B. (Eds), *Testy przesiewowe u dzieci i młodzieży w wieku szkolnym*. Instytut Matki i Dziecka. Warszawa 2002: 55–64.

Wożakowska-Kapłon B. Kempkiewicz T., Biskup P.: Serce sportowca. *Studia Medyczne Akademii Świętokrzyskiej* 2006; 4: 83–92.

*Wstępny ramowy projekt Narodowego Programu Zdrowia na lata 2006–2015. Ministerstwo Zdrowia, Państwowy Zakład Higieny*. Warszawa 2006. Available at: http://www.bpz.gov.pl/dokumenty/proj_npz_2006_15_11102005.pdf (14.06.2011).

Julia Durzyńska, Joanna Pacholska-Bogalska,
Anna Goździcka-Józefiak

# Epidemiological study of herpesviruses type 1 (HSV1) and type II (HSV II), cytomegalovirus (CMV) and human papillomavirus (HPV) in adolescent population by PCR method

**Abstract:** The purpose of the present study was to investigate the prevalence and genotypes of human papilloma viruses (HPVs) in the oral cavity of children and adolescents in the Wielkopolska region, Poland. The parallel aim of the present study was to elaborate a rapid, effective and time-saving multiplex PCR (mPCR) for the identification of oral herpetic infections in school children and adolescents. Our data provide novel information still missing in Poland (Levy-Bruhlet al., 2009) about the occurrence of the following viruses HPV, HSV-1, HSV-2 and HCMV in the oral cavity/oropharynx in young Polish population, aged 10–18.

The mPCR method has been conceptualized mainly for population-based noninvasive studies where a large number of probes are to be analysed and it can be concluded that it is accurate for DNA detection of HSV-1, HSV-2 and HCMV, thereby the mPCR brings to an end the entire processing cycle of clinical samples.

In molecular tests for the presence of HPV, HSV-1, HSV-2 and HCMV in the oral cavity, 3% of the studied population (125/4167 individuals) were found to be carriers of said viruses. Herpesviruses were twice as prevalent as HPVs. Genotyping of HPV proved HPV-11 to be the predominant genotype (39/46 positive cases), with other genotypes occurring much less frequently [HPV6 (3/46), HPV57 (3/46)], and HPV12 found in one sample only. It was found that virus carrier-state is not correlated with gender, neither clear correlation was found between the presence of DNA viruses and and the age of children and adolescence under study. Certain localities were identified as foci of infections, with more HPV, HSV-1, HSV-2, HCMV carrier cases observed.

**Key words:** multiplex PCR (mPCR), oral squamous cells, herpes infection, HPV infection, adolescents

## Introduction

Herpesviruses and papillomavirus represent a group of pathogens transmitted by close body contact and sexual relation. They can also be conveyed by body fluids and secretions or passed to a newborn by an infected mother. Those factors cause a number of diseases generally referred to as STDs (sexually transmitted diseases).

The group of sexually transmitted pathogens comprises also: chlamydia, gonorrhea, syphilis, trichomoniasis, Hepatitis B virus (HBV), and HIV virus. Despite many efforts to prevent STDs from spreading, the number of infections is constantly growing. Each year, approximately 19 million people around the world are estimated to be affected with STD, half of them between 15 and 24 years of age (source: Center for Disease Control and Prevention). STD incidence differs across races. It is highest among Black and American Indian/Alaska (approx. 3.7%), Asians (0.2%). In Europe, about 2% of the population suffer from STDs. Some types of STDs may give rise to serious complications, particularly with women [Ressel 2002]. They often lead to infertility and certain types of neoplasms. Most STDs tend to show no symptoms or may develop in various parts of the body, manifesting themselves clearly with irregular discharge, a rash or bump. Early detection of factors responsible for infections and preventive measures are important ways to check the spread of STDs. Owing to the development of molecular biology, many pathogens occurring in a low number of copies can now be identified in swabs collected from various parts of the body. Particularly useful for HSV and HPV analyses are swabs and lavage fluid from the oral cavity and genital organs. Infection analysis in swabs from the oral cavity is a non-invasive and convenient method, particularly useful in screening tests for children and school adolescents.

HSVs and HPVs exhibit a particular tropism for epithelial cells of the oral cavity and genital organs.

The herpesviridae family, contains more than 100 vertebrate viruses, and is one of the oldest family of DNA viruses known to infect human. Herpesviridae have spherical, enveloped and pleomorphic virion, 150–200 nm in diameter. The genom consists of doubled stranded DNA (dsDNA) and contains from $96\times10^6$ to $145\times10^6$ kilobase pairs with terminal and internal repeated sequences. The HSV genome encodes approximately 100 proteins. The half of the proteins are required for viral replication, the others facilitate the virus's interaction with different host cells or elicit the immune response. HSVs encode at least 11 glycoproteins that serve as viral attachment proteins (gB,gC, gD, gH), fusion proteins (gB), structural proteins, immune escape proteins (gC, gE, gI) and enzymes, including a DNA-dependent DNA polymerase, deoxiribonuclease, thymidine kinase, ribonucletide reductase, protease and other functions (nucleotidylyted phosphoprotein – ICPO, immediate early protein UL123, IE1, tegument protein-p65). HSVs can infect most types of human cells and even cells of other species. Herpesviruses can cause lytic infections of fibroblasts and epithelial cells, persistent and latent infections of neurons and Epstein-Barr virus immortalizing infections. DNA replication and assembly occur in the nucleus, virus buds from nucleus membrane and is released by exocytosis and cell lysis.

Human herpesviruses, or herpes simplex virus, is one of the most common agents infecting humans of all ages. The virus occurs worldwide and produces various illnesses, including mucocutaneous infection, infection of the CNS (Central nervous system), and occasionally infection of the visceral organs, infections during childhood and adolescence, and serious disease in individuals who are immunocompromised.

The are 8 herpesviruses groups known to infect humans.

I. Herpes simplex virus I (genus: simplexvirus).
II. Herpes simplex virus II (genus: simplexvirus).
III. Varicella zoster virus (genus: voricellovirus).
IV. Epstein-Barr virus (genus: lymphocryptovirus).
V. Cytomegalovirus (genus; cytomegalovirus).
VI. Human herpesvirus 6 (genus:roselovirus).
VII. Human herpesvirus 7 (genus: roseolovirus).
VIII. Human herpesvirus 8 (genus:rhadinovirus) [Murrey et al. 2002].

HSV-1 is the most common herpes simplex virus and most people develop it in childhood. HSV can cause lesions around the mouth and lips, such as cold sores (fever blisters), or infection of the eye. HSV is transmitted by such forms of contact as kissing an infected person or sharing eating utensils, towels, or razors. A person with a cold sore who performs oral sex with an another person can give that person genital lesions with HSV-1. Sores may develop as late as 20 days after exposure to the virus. Once the virus enters your body, it may emerge years later near the original site entry. About two days before an attack you may experience itching or sensitivity at the site. The virus may be triggered by certain foods, stress, fever, colds, allergens, sunburn, and menstruation. Almost 90% of people will have antibodies to HSV-1 by the time they reach adulthood. The CDC estimates one in four people are infected with herpes simplex virus 2 (HSV-2). HSV2 is transmitted through sexual contact and skin to skin contact. It is very contagious and can be contracted during sexual activity even if you don't have an open sore. Genital herpes simplex virus infection in older adolescents and adults is a major public health problem, having markedly increased in prevalence in the last 3 decades. This increased prevalence of genital herpes simplex virus infections poses major threats to newborns because most infections in neonates are acquired perinatally. Neonatal herpes simplex virus infection is a disease with high morbidity and mortality rates [Muller et al. 2010].

HSV-2 can affect the brain and central nervous system. The disease herpes encephalitis occurs when a latent HSV-2 virus is activated. Untreated herpes encephalitis is fatal in over 80% of cases. Respiratory arrest can occur within the first 24–72 hours. Fortunately, rapid diagnostic tests and treatment with acyclovir have significantly improved both survival rates (up to about 80%) and reduced complication rates (to nearly 40%). For those who recover, nearly all suffer some impairment, ranging from very mild neurological changes to paralysis. Recovery from HSV encephalitis depends on the patient's age, duration of diseases, the promptness of treatment

Herpes meningitis, an inflammation of the membranes that line the brain and spine cord, occurs in up to 10% of cases of primary genital HSV-2. Women are at

higher risk for herpes meningitis than men are. Symptoms include headache, fever, stiff neck, vomiting, and sensitivity to light. Fortunately, herpes meningitis usually resolves without complications, lasting for only 2–7 days, although recurrences have been reported. Published data shown that 45 million people aged 12 years or older have HSV-2 antibodies.

## Cytomegalovirus CMV

Cytomegalovirus is a common human pathogen, infecting 0.5% to 2.5% of all newborn and approximately 50% of the adult population in developed countries.

CMV has the largest genome of the human herpesviruses. The virus replicates only in human cells: fibroblasts, epithelial cells, macrophages, and other cells are permissive for CMV replication. Therefore, cytomegalovirus is a member of a group of herpes type, virus that can cause disease in different parts of the body in people with weakened immune system. CMV is particulary important as an opportunistic pathogen in immunocompromised patients. Once a person had a CMV infection, the virus usually lies dormant (or inactive) in the body, but it can be reactivated- and cause serious illness. The symptoms of a CMV infection vary depending upon the age and health of the infected person, and how the infection occurred. Infants who are infected before birth usually show no symptoms of a CMV infection after they are born, although some of them can develop hearing, vision, neurological, and developmental problems over time. In a few cases, there are symptoms at birth, which can include premature delivery, being small for gestational age, jaundice, enlarged liver and spleen. These infant are also at high risk of developing hearing, vision, neurological, and developmental problems. CMV is passed from person to person through close contact with body fluids, such as saliva, semen, vaginal fluids, blood, urine, tears and breast milk. Newborns can also contract CMV infection during or soon after birth by passing through the birth canal of an infected mother, consuming breast milk from a mother with the virus, or receiving a transfusion of blood donated by a person infected with CMV. Most of these infants show no symptoms of CMV infection: however, a few may develop pneumonia or other symptoms [Muller et al. 2010].

The burden of HSV infections is great and includes both physiological and psychosocial morbidity. HSV appears to be an important public health problem from the health economic point of view. In the last decade, a particular interest has been focused on HPVs as important factors in etilogy of many types of cancer.

## HPV

Papillomaviruses are epitheliotropic viruses present in the skin and mucosa of many animals. In humans, more than 100 types HPVs have been described. Mucosal and genital HPVs consisting of about 30 types, are divided into low-risk (HPV 6, 11, 40, 42, 43, 44, 54, 61, 70, 72, 81) and high-risk (HPV16, 18, 31, 33, 35,

39, 45, 51, 52, 56, 58, 66, 73, 82), according to their presence in malignant lesions of the cervix.

All HPVs are small circular double-stranded DNA viruses of about 8kbp. The genom can be subdivided into three parts, an early region containing early genes (E1-E8), a late region (L1 and L2 coding structural proteins), and a long control region (LCR) in which are located the binding elements for viral and cellular transcription factors. The E6 and E7 proteins of HPV-16 and HPV-18 have been identified as oncogenes, because they bind and inactivate the cellular growth-suppressor proteins, p53 and RB. E6 binds to p53 protein and targets it for degradation, and E7 binds and inactivates RB. These viruses can cause lytic infections in permissive cells but cause abortive, persistent, or latent infections or immortalize (transform) nonpermissive cells.

Only a small fraction of women infected with high risk HPV types eventually progressed to higher grade intraepithelial lesions and cervical cancer. The studies indicated that the risk for disease progression seems to be associated with the persistence of infection with high-risk HPV types, and with the amount of virus present, the viral burden. The other factors, such as diet, oral contraceptive use, tobacco smoking, co-infection with human immunodeficiency virus or other sexually transmitted diseases are also important.

The 70% of cervical cancer cases result from infection with HPV16 and 18, 89% from HPV 16, 18, 45, 31 and 33. The 87% of cervical adenocarcinoma result from infectioon with HPV 16, 18 and 45. HPV16 and 18 are also responsible for a significant fraction of cancer of the vulva (40%), vagina (80%), penis (40%), the oral cavity (25%) and the oropharynx (35%). HPV6 and 11 are responsible for 90% of genital warts and RRP (ang. recurrent respiratory papillomatosis) [Handisurya et al. 2009].

Worldwide estimates on the burden of HPV indicated that 300 million infections has no abnormality, 30 millon genital warts, 30 million Low-grade Cervical Lesions, 10 million High-grade Cervical Lesions and 0.5 million cervical cancer, genital cancer, and other. Thus, early screening and treatment of HPV infection together with education campaigns concerning lifestyle factors, may well decrease the risk of intraepithelial lesions developing into cervical cancer.

## Infection identification and prevention

Laboratory diagnosis of HSV and HPV infections involves:
- direct microscopic examination of cells from base of lesion,
- assay of tissue biopsy, smear or vasicular fluid for viral antigen,
- cell culture only for HSVs,
- serology and DNA probe analysis.

Considering the prevalence of HPV and HSV infections in humans and their relationship with many diseases, the recent decade has seen growing efforts to develop an effective vaccine against those viruses as a priority of health care around the world. In the absence of an appropriate cell line to multiply HPVs, the acquisi-

tion of vaccines from attenuated viral particles was impossible. To this end, however, methods of molecular biology and biotechnology were used.

Up to date, two preventive, clinically tested HPV vaccines HPV have been developed and marketed. A quadrivalent vaccine contains four types of virus-like particles (VLP) which are made of the main L1 capsid protein [Bishop et al. 2007] and for types of HPV viruses (HPV-6, -11, -16, -18) [Huang 2008]. A divalent vaccine contains two types of virus-like particles (HPV-16 and HPV18) [Jenkins 2008]. Capsid protein is the main immunogene of HPVs [Bishop et al. 2007]. If administered at proper dose and conformation, it induces immunity that can be sustained for many years, provided the antigen has been delivered according to manufacturer's specifications at doses duly divided (e.g. into 0, 2 and 6 months). In line with Europe's and world's coordinated vaccination programme guidelines (still missing in Poland) [King et al. 2008], the vaccine is most recommended for girls and young women before sexual initiation. Some experts claim that girls should be vaccinated already at the age of nine. It is well-known that HPV prevalence in this age group is very low. Very few countries, e.g. Austria, have included male adolescents into their obligatory vaccination programmes [Bosch et al. 2008].

As we know, many types of HPVs produce infection in humans, with over 30 of them threatening to contaminate cervical epithelium. Studies on cross-immunity against HPV genotypes have shown the immunity to be sufficient under the condition that the viruses are phylogenetically related to one another [Harper et al. 2006].

Virus-like particles used to produce vaccines are generated in yeast cells (*Saccharomyces cerevisiae*) or insect cells (*Spodoptera frugiperda Sf-9, Trichoplusia ni Hi-5*). The main L1 capsid protein of HPVs is capable of spontaneously assembling into virus-like particles in eukaryotic cells. Purification of VLPs is performed by the same methods as those used to purify infectious HPV virions from virus-infected cells (e.g. centrifugation in sacharose gradient) [Kirnbauer 1992]. Virus-like particles can also be generated in prokaryotic cells (*Escherichia coli*), where the main capsid protein occurs primarily in the form of capsomers. They need to be purified from a protein extract, e.g. by the method of chromatography, and then 'forced' to assemble into virus-like particles in *in vitro* conditions. One of the prerequisites for this process to occur is that the protein is incubated in a solution of high ionic strength (NaCl present). Serological studies have shown that virus-like particles produced this way have a full spectrum of immunogenic properties [Chen 2001].

Preventive vaccines do not affect regression of neoplastic lesions. Therefore, for many years therapeutic solutions have also been sought, which represents a much greater challenge. Works on curative and preventive vaccines were started at about the same time, but none of the former type have as yet been approved for commercial marketing. The main difference being that preventive vaccines induce humoral response in healthy persons (assumed to be HPV negative), while the removal of cells whose transformation has been caused by HPV in actively infected persons requires a strong T-lymphocyte stimulation. For that reason, treatment of existing lesions (CIN2-3, cervical cancer) concentrates on HPV oncogenic proteins: E6 i E7. Many pharmaceutical companies have taken efforts to develop E6 and E7 proteins

of the type inducing cellular response, capable of combating pre-neoplastic lesions. A number of strategies have been worked out to achieve that objective: Modified Vaccinia Ankara (MVA) virus, fusion proteins, peptides, bacterial DNA vectors (plasmids) and eukaryotic DNA vectors were all tried. An interesting strategy is to apply heat shock protein (HSP) in combination with E7 protein. In this solution, dendritic cells will be the first to be activated to present the E7 viral antigen to T lymphocites. Similar studies are being carried out at Arkansas University, their purpose being to introduce the full length of the E7 HPV16 and HPV18 protein into autologic dendritic cells. A promising idea is to use virus-like particles, as in preventive vaccines, with E7 protein attached to their surface. This strategy was developed by Ian Frazer, Queensland University. According to the latest research, an important role in immunological response to HPV infection is played by Langerhans cells, which are used by human papillomavirus to get out of the host immunity system.

While preventive HPV vaccination are commonly applied, it is still essential that epidemiological examinations are performed, particularly with children and adolescents. The diversity in the HPV genotypes and frequency of occurrence in healthy individuals from various populations have already been described.

We know, for example, that representatives of some populations are more likely to have their oral cavities and throats infected with high risk HPVs [Smith et al. 2007], while others are more frequently affected with low risk HPVs [Kurose et al. 2004]. Infections with high risk HPVs may lead to neoplastic lesions of the neck and head, which constitute a major health issue worldwide [Gillson 2008]. It is estimated that in the USA alone the total number of new HPV infections exceeds 6 million a year (National Cancer Institute). Studies conducted on material from genital tract show that in Europe prevalence of high risk HPV infections is highest among young women and relatively low among middle-aged women [De Vuyst et al. 2009]. It has been demonstrated that Poland is a leading EU country in terms of incidence of cervical cancer, which is an effect of persistent HPV infection. The number of registered cervical cancer cases in Polish population is around three times larger (17/100,000 – mean for Cracow and Warsaw) than in West European countries. These results rank Poland at the level of some South American countries and India [Bardin et al. 2008].

It seems, that a person should be checked for HPV presence before being given a preventive vaccine. If found HPV positive, a closer examination should be performed involving the establishment of HPV genotype and serostatus of the person concerned. An ideal solution, hopefully in the near future, would be to carry out preventive vaccination of uninfected children and a preventive and therapeutic vaccination for persons already infected with a specific type of HPV. This strategy will prevent HPV infections from occurring in populations covered by vaccination schemes.

Studies on herpesvirus vaccine have been done for many years. First vaccines were based on strains attenuated by multiple passage of the virus in cell cultures (full protection in humans is observed only for VZV); inactivation of the entire virus; administration of envelope glycoprotein and other genetically engineered viral

antigens; as well as by the introduction of DNA carriers (vectors) enabling viral antigen expression in target cells. Among attenuated vaccines was the strain of HSV-1 referred to as HSV-1 NS-gEnull. This virus had a deletion in the E glycoprotein gene, which largely restricted its cell penetration capability, while its antigen properties were preserved [Brittle 2008]. A HSV-2 strain was also generated with a deletion of the H glycoprotein encoding sequence, enabling the cell-to-cell infection propagation. The weakness of this vaccine is in its failure to address the risk of virus reactivation in infected patients. A strain of promising properties in terms of vaccine production is being developed by William Halford's team. The researcher assumed that if a living attenuated VZV (Oka strain) works as an excellent vaccine, there must be a way to apply an analogous solution to generate vaccines against HCV-1 (and subsequently also against HSV-2). A HSV-1 (HSV-1 *ICP0* mutant) was developed, which bears a mutation in the ICP (infected cell protein) region, which is responsible for keeping the balance between latency and replication of viral DNA. Such a modified HSV-1 is highly susceptible to repression induced by alpha and beta interferon, is non-virulent in immunocompetent mice and provides a very high response to the infection with wild HSV-1, causing it to be almost entirely removed [Halford et al. 2010]. It appears, then, that HSV-1 ICP0 and HSV-2 ICP0 mutants may be an excellent choice for massive vaccination against *herpes genitalis* and *herpes labialis*.

A HCMV (Towne line) strain has also been produced by multiple passage in WI-138 fibroblasts, free of virulence but showing antigen activity. In vaccines containing this virus, the immunological response proved to be several times weaker than that induced by a wild strain [Jacobson 2009].

Genetic engineering techniques have also been employed in the production of HSV vaccine. In this case, viral antigens were produced in cell lines (e.g. hamster's oocytes). Vaccines obtained this way include the one comprising B glycoprotein which, being deprived of transmembrane domain, was secreted out of the cell. It turned out, however, that the induction of humoral response was to weak to sufficiently prevent the virus from being reactivated from the state of latency. Yet another method to produce vaccines is the alphavirus replicon system, which generates virus-like replicon particles (VRPs) containing pp65 (ppUL83) protein of HCMV or IE1 (UL123) protein. A vaccine model combining gB, pp65 and IE1 proteins has also been created [Schleiss et al. 2010].

Others are based on plasmids, which may serve as carriers of open reading frames for various viral proteins with strong immunogenic effects. An open reading frame, e.g. for glycoprotein gD, may be modified to ensure that the protein is secreted out of the cell, where it can be recognised by the immunity system – B limphocytes, producing immunoglobulins IgG1 and IgG2, and T limphocytes are involved in the cellular response. Unfortunately, even these types of vaccines fail to provide full protection against infection/secondary infection [Ferenczy 2007].

Up to this day, there is no single vaccine against HSV-1 and HSV-2 to effectively prevent infection from recurring. The only one marketed for many years is VZV (varicella-zoster virus, human herpes virus 3, HHV-3) protecting against chickenpox and zoster infections. The vaccine was obtained by multiple passage of the vi-

rus collected from a carrier, and the strain thus received is known as Oka. Oka strains are characterised by reduced IE62, 65, 66 and 67 gene transcription, which prevents the virus from spreading from cell to cell. The Oka line is also marked with a diminished production of glycoprotein I (IE 67gene), which occurs in abundance on the viral surface of wild strains. The task of the protein I is to form heterodimer with gE glycoprotein and bind immunoglobulin to enable cell-to-cell transfer of VZV virions [Cohrs et al. 2006].

EBV vaccines (human herpes virus 4) are produced using similar strategies, as those applied for other Herpesviridae. Of particular note in this case is gp350 glycoprotein, which – combined with an adjuvant – has delivered very promising results both at the level of humoral and cellular response, passing successfully phase I and II clinical tests. Other attempts have also been made to create EBV vaccine involving the synthesised fragments of virus nuclear antigens, e.g. EBNA3 (Epstein-Barr nuclear antigen). Those peptides have been coupled with an adjuvant and inactivated tetanus anatoxin. It seems that the efficacy of the vaccine may additionally be improved with tetanus anatoxin replaced with gp350 glycoprotein.

An early identification of herpetic infections in human population is important since efficient herpetic prophylaxis has not yet been developed, and a number of investigations are performed on anti-herpetic treatments and vaccines [Wilson et al. 2009]. It is assumed that during active infections (primary or recurrent), herpes viruses (especially HSV-1, HSV-2 and HCMV) are abundantly shed in the oral cavity and their genomic DNA may be detected in the total DNA isolated from oral squamous cells; therefore it is conceivable that serostatus of an individual is not assessed.

## Aim of the study

It should be noted that there are very few population-based screening programmes restricted to small areas in Poland [Bardin et al. 2008]. The purpose of the present study was to investigate the prevalence and genotypes of HPV in the oral cavity of children and adolescents in the Wielkopolska region, Poland. It is the first epidomiological cross-sectional survey conducted in children and adolescents in Poland. The parallel aim of the present study was to elaborate a rapid, effective and time-saving multiplex PCR for the identification of oral herpetic infections in school children and adolescents. Our data provide novel information still missing in Poland [Levy-Bruhl et al. 2009] about the occurrence of the following viruses HPV, HSV-1, HSV-2 and HCMV in the oral cavity/oropharynx in young Polish population, aged 10–18.

# Material and methods

## Sampling design

Data were collected in years 2008–2009 as part of a large ongoing cross-sectional population-based survey of adolescents in the Wielkopolska region named ADOPOLNOR (www.adopolnor.eu). This region with 3.4 mln of population is situated in the central west of Poland and covers 29,826.51 sq. km. The study population consisted of 2040 boys and 2127 girls aged 10–18 years, pupils of all types of schools, residing in rural and urban areas of the Wielkopolska region. The examination included body height and weight measurements and an extensive questionnaire where adolescents reported their state of health, health-risk behaviours, such as smoking cigarettes, drinking alcohol, taking drugs and practicing risky sexual activities, and socio-economic life conditions of their households.

## Biological samples – collection and preparation

Oral squamous cells from all the studied subjects (n=4167) were transported frozen (in a portable freezer) as swabs and oral rinses from schools, where they were collected, for immediate laboratory processing. Buccal cells from swabs were dispersed into bijoux bottles already containing 5ml of buccal saline solution (oral rinse from the same individual) in order to enrich its content of nucleated squamous cells. They were observed under microscope, centrifuged (10 min, 3,000 rpm), and resuspended in 200 μl of Proteinase K digestion buffer; total DNA isolation from oral squamous cells was performed with a ready-to-use set, A&A Biotechnology Swab (cat. nr 025–100, A&A Biotechnology, Gdynia, Poland). Before further analysis, the quality of the total cellular DNA was verified by a PCR amplification of a c-fos fragment [Durzyńska et al. 2011]. DNA concentration obtained from buccal swabs ranged from 10 to 30 ng/ul.

## Detection of HPV DNA by MY-PCR, sequencing and preparation of DNA standards

Total DNA extracted from oral squamous cells was used as a template in MY-PCR amplification with MY09 and MY11 degenerate oligonucleotides complementary to the HPV-L1 open reading frame, allowing for detection of 33 HPV genotypes. All MY-PCR reactions were performed in the Biometra T-gradient thermocycler (Biometra GmbH, Goettingen, Germany) using the following procedure: (1) incubation with UNG at 55°C for 2 min; (2) pre-denaturing at 95°C for 5 min; (3) annealing of oligonucleotides at 55°C for 30 sec; (4) elongation at 72°C for 45 sec; (5) denaturing at 95°C for 30 sec; (6) final elongation at 72°C for 7 min. Steps 3–5 were repeated 40 times. Reaction volume was 10 μL, containing 1 mM of each oligonucleotide, 0.6mM MgCl2, 1xKCl buffer for Taq polymerase, 0.4U of Taq polymerase (Fermentas International, Burlington, Ontario, Canada), 0.1U of UNG

(Jena Bioscience, Jena, Germany), and 2mM of each dNTP (dATP, dTTP, dGTP, dCTP, and UTP). For diagnostic MY-PCR 20–50 ng of total DNA from all individuals (2 $\mu$L) were used, and 5 mL of MY-PCR products were electrophoresed in 1.5% agarose gel (Prona Agarose, Madrid, Spain) containing ethidium bromide. All positive PCR products containing 450-bp HPV PCR products were subjected to direct DNA sequencing in order to determine HPV genotype. MY-PCR samples containing visible bands were directly sequenced using 1–2 $\mu$L of the MY-PCR reaction, and 50 pmol of the sequencing oligonucleotide (both MY09 and MY11 in two separate sequencing procedures). Sequencing was performed with BigDye Terminator v3.1 on an ABI Prism 3130XL Analyzer (Applied Biosystems, Foster City, CA). Sequence chromatograms were checked for accuracy, and contigs were edited and assembled using FinchTV 1.3.1 (Geospiza Inc., Seattle, WA), and GenDoc 2.7.000. The nucleotide sequences were compared with GenBank sequences using the BLAST program. In each MY-PCR round an appropriate MY-PCR control was processed concurrently (1): a positive control, a plasmid containing the HPV11-L1 open reading frame produced for our prior studies; and two plasmids containing HPV-L1 open reading frames from HPV16 and HPV18 (kindly provided by G. Zur Hausen and Marco Ciotti, respectively) were combined in equimolar proportion at an approximate final plasmid DNA concentration of ~1 pg/$\mu$L. Then human cellular HPV-negative DNA (~20 ng/$\mu$L) was added to the mixture of plasmids in a 1:1 volume ratio, and 2 mL was used as a positive control. Negative controls were water and/or HPV-negative samples. Strict precautions were taken in order to avoid falsepositive results, and good laboratory practice was implemented: (1) one-direction sample processing using separate rooms for DNA isolation, preparation of MY-PCR, thermal cycling, and electrophoresis; (2) UNG and dUTP addition to the MY-PCR reaction mix with the use of filter tips; (3) decontamination of laboratory equipment with solutions that degrade DNA; and (4) the purity of water and other reagents was regularly verified [Durzyńska et al. 2010a, Durzyńska et al. 2010b].

## Preparation of herpes controls and detection of herpes DNA by multiplex PCR

HSV-1, HSV-2, and HCMV genomic sequences were retrieved from the database at http://www.ncbi.nlm.nih.gov/Taxonomy/Browser and submitted for bioinformatic analysis (Primer3, a software tool available online http://frodo.wi.mit.edu/ primer3/) in order to select oligonucleotides for amplification of DNA fragments of different length of each virus. Many pairs of oligonucleotides were selected and synthesized at the Institute of Biochemistry and Biophysics, Warsaw, Poland (http://www.ibb.waw.pl/). All pairs of oligonucleotides were tested, and finally three pairs amplifying herpetic target sequences under the same thermal conditions were assayed in a mPCR (Table 1); a HSV-1 DNA amplicon of 333 bp length, a HSV-2 DNA amplicon of 212 bp length, and a HCMV DNA amplicon of 486 bp length were obtained using ATCC herpes DNA standards as a template (American Type Culture Collection, www.atcc.org). All three amplicons were purified (PCR purification kit, Qiagen, Venlo, Netherlands), and cloned into a

Table 1. Pairs of specific oligonucleotides used for herpes viral DNA amplification in Multiplex PCR

| Name of the starter | Oligonucleotide sequence 5'→3' | The length of the amplified fragment |
|---|---|---|
| HSV-1for | CGTGATTTTGTTTGTCGTCATAG | 333 bp fragment of a human herpesvirus 1 Strain KOSc(C2), glycoprotein D (US6) gene |
| HSV-1back | GTCAGGTTGTAGGGTTGTTTCC | |
| HSV2-3 | CTAGTTGTCGCGGTGGGACT | 212 bp fragment of a human herpesvirus 2 Strain MS, glycoprotein D gene |
| HSV2-4 | TAGTACACAGTGATCGGGATGC | |
| HCMVfor | TCTATCTGGAGATGCTGCTGAA | 486 bp fragment of a human herpesvirus 5, Isolate I-39, UL56 (UL56) gene |
| HCMVback | TCTGGATATTCACATCGGACTG | |

pGEM®-T Easy vector in separate reactions for each amplicon (Promega Corporation, Fitchburg, WI). Ligation of viral DNA amplicons was performed in the final volume of 12 $\mu$l in the following way: 6 ml of 2× rapid ligation buffer for T4 DNA ligase, 1 $\mu$l of pGEM1-T Easy vector (50 ng), 4 $\mu$l of purifiedPCRproduct, and 1 $\mu$l of T4 DNAligase (3 Weiss units/ml) were mixed and incubated at room temperature for 1 hr. Following transformation of *E. coli* cells, the plasmid DNA was isolated from bacteria using minipreparation kit (Qiagen) and submitted for DNA sequencing in order to confirm the insertion of viral amplicons. DNA sequencing was performed with BigDye Terminator v3.1 on an ABI Prism 3130XL Analyzer (Applied Biosystems, Foster City, CA). Sequence chromatograms were checked for accuracy, contigs edited, then assembled using FinchTV 1.3.1 (Geospiza Inc., Seattle, WA) and GenDoc 2.7.000 [Nicholas and Nicholas, 1997]. The nucleotide sequences were compared with GenBank sequences by using the BLAST program.

All PCR reactions were performed in Biometra T-gradient thermocycler under the following conditions: (1) incubation with UNG (uracil N-glycosylase): 55°C for 2 min, (2) pre-denaturation: 95°C for 5 min, (3) denaturation: 95°C for 30 sec, (4) annealing of oligonucleotides: 55°C for 30 sec, (5) elongation: 72°C for 30 sec, and (6) final elongation: 72°C for 7 min. Steps 3–5 were repeated 40 times. Reaction volume was 10 ml containing: 1 mM of each oligonucleotide (see Table 1), 0.6mM MgCl2, 1× KCl buffer for Taq polymerase, 0.4U of Taq polymerase (Fermentas International, Burlington, Canada), 0.1U of UNG (Jena Bioscience, Jena, Germany), 2mM of each dNTP (dATP, dTTP, dGTP, dCTP, dUTP). While setting up PCR conditions and preparing positive plasmid controls, about 10 ng of total DNA from cells infected with HSV-1, HSV-2, and HCMV (ATCC standards) were used. Plasmids containing all the respective viral inserts were prepared for DNA sequencing at the concentration of ~100 ng/ml (as determined by spectrophotometric measurement), then they were mixed in equimolar amounts and diluted serially by factor of 10 in order to determine the minimal detectable copy number of viral DNA. It was calculated using the Avogadro constant, molecular masses of pGEM1-T Easy plasmid and viral inserts, that 1 ml of the initial mix of plasmid constructs contained approximately $1 \times 10^{10}$ copies of each plasmid. Subsequently, 2 μl of a dilution containing approximately 100 copies were used as a posi-

tive control for eachmPCRdiagnostic series of reaction. In order to imitate chemical conditions of DNA isolates from clinical samples, 20 ng of human herpes-free genomic DNA were added to the control plasmid template. About 20–50 ng (2 $\mu$l) of the total cellular DNA isolated from clinical samples were used for screening in mPCR reactions. In order to avoid false positive results, good laboratory practices were implemented, such as: (1) one direction sample processing—separate rooms used for DNA isolation, preparation of PCR reaction, thermal cycling, and electrophoresis, (2) UNG and dUTP were added to the PCR reaction mix and filter tips used, (3) and laboratory equipment and benches were decontaminated with solutions for DNA degradation, and (4) purity of water and other reagents was verified every now and then [Durzyńska et al. 2011].

## Results

N=4167 children and adolescents participated in the study of HPV, HSV-1, HSV-2 and HCMV carriage in the oral cavity, with boys accounting for 48.96% of the study group and girls representing 51.04%. The group was further subdivided into primary, lower secondary and higher secondary school categories. Social and economic diversity and sexual initiation were also taken into consideration. Detailed data are shown in Table 2.

In molecular tests for the presence of HPV, HSV-1, HSV-2 and HCMV in the oral cavity, 3% of the studied population (125/4167 individuals) were found to be carriers of said viruses. Herpesviruses were twice as prevalent as HPVs. Detailed data are shown in Table 3. Genotyping of HPV proved HPV-11 to be the predominant genotype (39/46 positive cases), with other genotypes occurring much less frequently [HPV6 (3/46), HPV57 (3/46)], and HPV12 found in one sample only. Detailed genotype analysis is shown in Table 4.

Correlation was examined between virus carrier-state and a number of variables, such as gender, age, place of residence and location of school. It was found that virus carrier-state is not correlated with gender (Table 5). No clear correlation was also found between the presence of DNA viruses and and the age of children and adolescence under study; although there is a several percent difference between subjects aged 13–14 showing the most virus positive cases (15.2 and 14.4%, respectively) and other age groups. Detailed data are shown in Table 6.

The relationship between the carrier-state of indicated viruses and place of residence is very complex. Our results show that there are twice as many virus carriers in rural areas than in other location categories, if two sub-categories are to be distinguished (other than rural areas): large cities (population of over 500,000) and smaller towns. However, with the division limited to two categories of urban areas (population of over 20,000) and rural areas (below 20,000), the percentage of carriers is very similar in both categories (Table 7). Certain localities were identified as foci of infections, with more HPV, HSV-1, HSV-2, HCMV carrier cases observed. Due to a much lower population, apart from Poznań, particular note should be

Table 2. General characteristics of the study sample

| Variables | | N | % |
|---|---|---|---|
| Gender | boys | 2040 | 48.96 |
| | girls | 2127 | 51.04 |
| Age | 10–12 (primary school) | 1318 | 31.63 |
| | 13–15 (lower secondary school) | 1558 | 37.39 |
| | 16–18 (higher secondary school) | 1291 | 30.98 |
| Place of residence* | village | 1613 | 38.71 |
| | town: population of 21,000–500,000 | 1685 | 40.44 |
| | city with population of over 500,000 | 827 | 19.85 |
| Socio-economic status (SES – on the basis of complex indicator) ** | low | 1343 | 32.23 |
| | average | 1311 | 31.46 |
| | high | 1406 | 33.74 |
| Sexual intercourse*** | no | 2356 | 86.52 |
| | yes, once | 103 | 3.78 |
| | yes, more then once | 264 | 9.69 |

* 42 individuals did not state their place of residence (blank space in the questionnaire)
** 107 individuals did not state their SES (blank space in the questionnaire)
***Only adolescents (13–15 and 16–18 age categories) were asked the question about sexual intercourse (N=2849). 126 individuals did not want to answer this question (blank space in the questionnaire), thus N=2723 in this category

taken of the cities like Turek or Zbąszyń where 35 and 17 cases were found respectively. Detailed list of correlations between carrier-state and shool location is shown in Table 8.

Table 3. Oral cavity carriers of viruses in the study sample

| Type of virus | Number of virus positive cases (n) | % of oral cavity carriers (N=4,167) |
|---|---|---|
| HPV | 46 | 1.10 |
| CMV | 34 | 0.82 |
| HSV-1 | 9 | 0.21 |
| HSV-2 | 36 | 0.87 |
| Total | 125 | 3.00 |

Table 4. Distribution of oral cavity HPV genotypes in the study sample

| HPV genotype | Number of HPV genotype positive cases (n) | % of carriers, oral cavity (compared to N=4,167) |
|---|---|---|
| HPV-11 | 39 | 0.94 |
| HPV-6 | 3 | 0.07 |
| HSV-57 | 3 | 0.07 |
| HSV-12 | 1 | 0.02 |
| Total | 46 | 1.1 |

Table 5. Oral cavity HPV, HSV-1, HSV-2 and HCMV carriers by gender

| Gender | n | % (of 4167) | % (of 125) |
|---|---|---|---|
| Boys | 63 | 1.51 | 50.4 |
| Girls | 62 | 1.49 | 49.6 |
| Total | 125 | 3.00 | 100.0 |

Table 6. Oral cavity HPV, HSV-1, HSV-2 and HCMV carriers in different age groups

| Age | n | % (of 4167) | % (of 125) |
|---|---|---|---|
| 10 | 14 | 0.34 | 11.2 |
| 11 | 12 | 0.29 | 9.6 |
| 12 | 10 | 0.24 | 8.0 |
| 13 | 19 | 0.46 | 15.2 |
| 14 | 18 | 0.43 | 14.4 |
| 15 | 12 | 0.29 | 9.6 |
| 16 | 13 | 0.31 | 10.4 |
| 17 | 15 | 0.36 | 12.0 |
| 18 | 10 | 0.24 | 8.0 |
| Total | 125 | 3.00 | 100.0 |

Table 7. Oral cavity HPV, HSV-1, HSV-2 and HCMV carriers by population (village, different categories of towns)

| Place of residence | n | % (z place of residence – Table 1) | % (of 125) |
|---|---|---|---|
| Village | 61 | 3.78 | 48.8 |
| Town 21,000–500,000 | 42 | 2.49 | 33.6 |
| Poznań | 22 | 2.66 | 17.6 |
| Total | 125 | 3.000 | 100.0 |

Table 8. Oral cavity HPV, HSV-1, HSV-2 and HCMV carriers by school location

| Location of school | n | % (of 4167) | % (of 125) |
|---|---|---|---|
| Biała | 1 | 0.024 | 0.8 |
| Kalisz | 6 | 0.144 | 4.8 |
| Krajenka | 5 | 0.120 | 4.0 |
| Lubasz | 7 | 0.168 | 5.6 |
| Opatówek | 2 | 0.048 | 1.6 |
| Osieczna | 7 | 0.168 | 5.6 |
| Piła | 5 | 0.120 | 4.0 |
| Poznań | 38 | 0.912 | 30.4 |
| Zbąszyń | 17 | 0.408 | 13.6 |
| Pięczkowo | 2 | 0.048 | 1.6 |
| Turek | 35 | 0.839 | 28.0 |
| Total | 125 | 3.000 | 100.0 |

## Discussion

A great variation in HPV rates detected in the normal oral mucosa was reported and it depends on the population studied and the choice of method. It is also known that HPV occurrence in the normal oral mucosa comprises sublinical and/or latent infections, morover HPV infections of low virus load are frequent in the oral cavity [Castro et al. 2006]. Given the abundance of literature describing results concerning HPV prevalence in the oral cavity and oropharynx all over the world, data presented in this study are to be compared to similar surveys, which were conducted on adolescents 10–18 years old using the same MY-PCR method.

In the present study HPV-11 genotype was detected at surprisingly high rate (39/46 of all positive cases), while HPV-6 type, frequently reported to be present in the oropharynx, was detected only in 3 cases (3/46). In our concurrent study, HPV-6 genotype has been identified in 30% cases of juvenile-onset recurrent respiratory papillomatosis among 47 patients investigated (3–12 year old) [Durzyńska et al. 2010a]. Concordant results concerning high HPV-11 prevalence in normal oral mucosa were not reported in literature. One should keep in mind that in some cases localized HPV-11 infection may lead to severe clinical symptoms: such as recurrent respiratory papillomatosis mentioned above or genital warts if infection is transmitted [Mammas et al. 2009]. Another important finding of our study was HPV-57 type present in three HPV positive samples (3/46). This HPV type had been previously found in numerous locations (inverted papillomas of the maxillary sinus, an oral verruca and a genital condyloma [Marquart et al. 2006]. In rare cases both HPV-11 and -57 types are detected in malignant tumors. HPV-11 is known especially for transformation of cells of larynx and lungs, while HPV-57 of nasal cavity.

It was also surprising to detect HPV-12 genotype in one specimen because in the majority of studies, it was not mentioned to infect buccal mucosa [Kojima et al. 2003; Syrjanen 2003] and is rather considered to be etiologically significant in cutaneous epidermodysplasia verrucifromis (EV)-associated HPV (EV-HPV). However, HPV-12 tends also to presistently infect within oral mucosa as shown in the study on HPV prevalence in Japan [Kurose et al. 2004].

In the present study HPV-HR genotypes were not detected, whereas their presence was reported at relatively high rate in similar studies by other researchers [Smith et al. 2007; Summersgill et al. 2001]. It may be due to the MY-PCR method solely employed in our experimental procedure. It has been reported that type-specific nested PCR greatly improves detection limit of HPV-16 in buccal samples, setting it below 10 viral DNA copies [Rice et al. 2000]. However, in one study performed with the sole usage of MY09 and MY11 oligonucleotides in PCR reaction, HPV-16 DNA was detected in one sample (among 662 screened). The detection limit was set at 100 viral copies in that study [Kurose et al. 2004]. These results could support ours, in which case indeed, HPV-HR types were not present in the study sample of Polish adolescents or HPV-HR DNA load was significantly lower (several viral copies) than the viral load of detected HPV-LR genotypes.

The overall HPV prevalence in the study group was 1.08%, and its distribution was even among different strata of age. This observation remains in opposition to those from other studies establishing that HPV prevalence depends strongly upon age [Smith et al. 2007]. Whereas, the HPV prevalence reported herein is low and consistent with results mendtioned above, where the mean HPV prevalence in the 12–15 year-old group of American adolescents was 1.5% [Smith et al. 2007].

In the present study correlations between HPV prevalence and other variables were not observed. Concordant results were described in a number of studies [Smith et al. 2007; Summersgill et al. 2001], where sociodemographic, gender, sexual intercourse (only adolescents over 13 years old were asked the question about their sexual activity), smoking cigarettes and drinking alcohol were taken into account. In our study correlation of HPV prevalence with upper respiratory chronic diseases (such as asthma, tuberculosis, mucoviscidose) was also sought but was not observed. However, it is noteworthy that in several schools, localized in small towns around Poznań and in Poznań itself, the number of HPV positive children/adolescents was elevated.

A non-invasive methods for oral squamous cells collection, DNA extraction and HPV DNA detection described herein are suitable for large cross-sectional studies of children and adolescents.

Development of molecular biology techniques has allowed identification of genetic material of pathogens contained in clinical samples. Virus propagation in cell cultures has become optional in many diagnostic procedures aiming at virus detection. In this study, a method for simultaneous detection of three herpes viruses, HSV-1, HSV-2 and HCMV, has been also elaborated. Similar approaches have already been applied in several investigations of herpetic DNA presence [Shin et al. 2003; McIver et al. 2005; Reszka et al. 2008]. In order to assess the accuracy of the multiplex PCR described herein a sample group of adolescents with varied characteristics was selected in Poland. The mPCR method has been conceptualized mainly for population-based noninvasive studies where a large number of probes are to be analysed and it can be concluded that it is accurate for DNA detection of HSV-1, HSV-2 and HCMV, thereby the mPCR brings to an end the entire processing cycle of clinical samples (swab and oral rinse collection, transportation, DNA isolation). Results presented here will be a guidance not only on the detection of infections with herpes viruses but also on the assessment of risky sexual behaviours.

The HSV-1 tends to infect the oral-labial area and is the source of a common cold sore. The closely related HSV-2 is the main cause of genital infection, a lifelong viral infection that is usually acquired through direct physical contact. HSV-1 can be transmitted from lips to genitals during oral-genital sex. Genital herpes is essentially an STD and rarely infects children before puberty. However, a child may be infected prenatally, perinatally or postnatally from his/her mother during the viraemia following maternal primary infection. About 10% of the population acquires HSV infection by the genital route and young adulthood is the most at-risk period for viral acquisition. For example, a Swedish study of 14–15 year-old girls indicated that 0.4% were HSV-2 seropositive, while 15 years later the number grew to 22% for the same study sample [Christenseon et al. 1992]. Viral transmission

also depends on sexual behaviour, socioeconomic status, stress, psychological discomfort, and local and systemic immune response to HSVs among sexual partners who transmit or do not transmit HSVs. At least 75% of the people infected with HSVs are asymptomatic during the first decade of life [Steben and Sacks, 1997]. HSV-1 infection of the oral cavity is common, with the source of virus usually being household or day-care contacts. Consequently, many children enter adolescence already infected with HSV [Stanberry and Roenthal, 1999]. HSV-1 and HSV-2 infections are common among adolescents. The prevalence of HSV infections in adolescents varies widely depending on geographic location and sexual behaviours [Lamey and Hyland et al. 1999].

Only 10% to 15% of adolescents are infected with HCMV, but this number increases to 50% with 35-year-olds [Schleiss 2009]. Transmission occurs via blood, organ transplantation and all secretions (urine, saliva, semen, cervical secretions, breast milk and tears). The majority of HCMV infections acquired in young adulthood are asymptomatic. HCMV is more prevalent among people of low socioeconomic status [Aynaud et al. 2002; Behzad-Behbanhani et al. 2008; Roizman and Sears 1996].

## References

Aynaud O., Poveda J.D., Huynh B., Guillemotonia A., Barasso R.: Frequency of herpes simplex virus, cytomegalovirus and human papillomavirus DNA in semen. *Int J STD AIDS* 2002; 13:547–550.

Bardin A., Vaccarella S., Clifford G.M., Lissowska J., Rekosz M., Bobkiewicz P., Kupryjanczyk J., Krynicki R., Jonska-Gmyrek J., Danska-Bidzinska A., Snijders P.J.F., Meijer C.J.L.M., Zatonski W., Franceschi S.: Human papillomavirus infection in women with and without cervical cancer in Warsaw, Poland. *Eur J Cancer* 2008; 44:557–564.

Behzad-Behbahani A., Entezam M., Mojiri A., Pouransari R., Rahsaz M., Banihashemi M., Heidari T., Farhadi A., Azarpira N., Yaghobi R., Jowkar Z., Ramzi M., Robati M.: Incidence of human herpes virus-6 and human cytomegalovirus infections in donated bone marrow and umbilical cord blood hematopoietic stem cells. *Indian J Med Microbiol* 2008; 26:252–255.

Bernard H.U., Calleja-Macias I.E., Dunn S.T.: Genome variation of human papillomavirus types: phylogenetic and medical implications. *Int J Cancer* 2006; 118(5):1071–1076.

Bishop B., Dasgupta J., Klein M., Garcea R.L., Christensen N.D., Zhao R., Chen X. S.: Crystal Structures of four types of Human Papillomavirus L1 Capsid Proteins. *Biol Chem* 2007; 282(43):31803–31811.

Bosch F.X., Castellsagué X., de Sanjosé S.: HPV and cervical cancer: screening or vaccination? *Br J Cancer* 2008; 98(1):15–21.

Brittle E.E., Wang F., Lubinski J.M., Bunte R.M., Friedman H.M.: A replication-competent, neuronal spread-defective, live attenuated herpes simplex virus type 1 vaccine. *J Virol* 2008; 82(17):8431–8441.

Castro T.P., Bussoloti Filho I.: Prevalence of human papillomavirus (HPV) in oral cavity and oropharynx. *Braz J Otorhinolaryngol* 2006; 72(2):272–282.

Chen X.S., Casini G., Harrison S.C., Garcea R.L.: Papillomavirus capsid protein expression in Escherichia coli: purification and assembly of HPV11 and HPV16 L1. *J Mol Biol* 2001; 307(1):173–182.

Christenseon B., Bottiger M., Svensson A., Jennssen S.: A 15-year surveillance study of antibodies to herpes simplex virus types 1 and 2 in a cohort of young girls. *J Infect* 1992; 25: 147–154.

Cohrs R.J., Gilden D.H., Gomi Y., Yamanishi K., Cohen J.I.: Comparison of virus transcription during lytic infection of the Oka parental and vaccine strains of Varicella-Zoster virus. *J Virol.* 2006; 80(5):2076–2082.

de Sanjosé S., Alemany L., Castellsagué X., Bosch F.X.: Human papillomavirus vaccines and vaccine implementation. *Womens Health* (*Lond Engl*) 2008; 4(6):595–604.

De Vuyst H., Clifford G., Franceschi S.: HPV infection in Europe. *Eur J Cancer* 2009; 45: 2632–2639.

Durzyńska J., Błażejewska P., Szydlowski J., Goździcka-Józefiak A.: Detection of anti-HPV11-L1 antibodies in immune sera from patients suffering from recurrent respiratory papillomatosis using ELISA. *Viral Immunol* 2010a; 23(4):415–423.

Durzyńska J., Pacholska-Bogalska J., Kaczmarek M., Durda M., Hanć T., Skrzypczak M., Goździcka-Józefiak A.: HPV genotypes in the oral cavity/oropharynx of children and adolescents: cross-sectional survey in Poland. *Eur J Pediatrics* 2010b; 170(6):757–761

Durzyńska J., Pacholska-Bogalska J., Kaczmarek M. et al.: Multiplex PCR for identification of herpes virus infections in adolescents. *J Med Virol* 2011; 83:267–271.

Elliott S.L., Suhrbier A., Miles J.J., Lawrence G., Pye S.J., Le T.T., Rosenstengel A., Nguyen T., Allworth A., Burrows S.R., Cox J., Pye D., Moss D.J., Bharadwaj M.: Phase I trial of a CD8+ T-cell peptide epitope-based vaccine for infectious mononucleosis. *J Virol.* 2008 Feb; 82(3):1448–1457. Epub 2007 Nov 21.

Ferenczy M.W.: Prophylactic vaccine strategies and the potential of therapeutic vaccines against herpes simplex virus. *Curr Pharm Des.* 2007; 13(19):1975–1988.

Gilson M.L.: Human papillomavirus-related diseases: oropharynx cancers and potential implications for adolescents HPV vaccination. *J Adolescent Health* 2008; 43:52–60.

Halford W.P., Püschel R., Rakowski B.: Herpes simplex virus 2 ICP0 mutant viruses are avirulent and immunogenic: implications for a genital herpes vaccine. *PLoS One.* 2010; 17, 5(8):12251.

Handisurya A., Schellenbacher C., Kimbauer R.: Disease caused by human papillomaviruses (HPV). *J. Dtsch. Dermatol Ges* 2009; 7:453–466.

Harper D.M., Franco E.L., Wheeler C.M., Moscicki A.B., Romanowski B., Roteli-Martins C.M., Jenkins D., Schuind A., Costa Clemens S.A., Dubin G.: HPV Vaccine Study group. Sustained efficacy up to 4.5 years of a bivalent L1 virus-like particle vaccine against human papillomavirus types 16 and 18: follow-up from a randomised control trial. *Lancet* 2006; 367: 1247–1255.

Huang C.M.: Human papillomavirus and vaccination. *Mayo Clin Proc* 2008; 83:701–706.

Jacobson M.A., Adler S.P., Sinclair E., Black D., Smith A., Chu A., Moss R.B., Wloch M.K.: A CMV DNA vaccine primes for memory immune responses to live-attenuated CMV (Towne strain). *Vaccine* 2009; 4;27(10):1540–1548.

Jenkins D.: A review of cross-protection against oncogenic HPV by an HPV-16/18 AS04-adjuvanted cervical cancer vaccine: Improtance of virological and clinical endpoints and implications for mass vaccination in cervical cancer prevention. *Gynecologic Oncology* 2008; 110:18–25.

King L.A., Lévy-Bruhl D., O'Flanagan D., Bacci S., Lopalco P.L., Kudjawu Y., Salmaso S.: Introduction of human papillomavirus (HPV) vaccination into national immunisation schedules in Europe: Results of the VENICE 2007 survey. VENICE Country Specific Gate Keepers and Contact Points. *Euro Surveill* 2008; 13(33): pii: 18954.

Kirnbauer R., Booy F., Cheng N., Lowy D.R., Schiller J.T.: Papillomavirus L1 major capsid protein self-assembles into virus-like particles that are highly immunogenic. *Proc Natl Acad Sci USA* 1992; 89(24):12180–12184.

Kojima A., Maeda H., Kurahashi N. et al.: Human papillomaviruses in the normal oral cavity of children in Japan. *Oral Oncol* 2003; 39(8):821–828.
Kurose K., Terai M., Soedarsono N. et al.: Low prevalence of HPV infection and its natural history in normal oral mucosa among volunteers on Miyako Island, Japan. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2004; 98(1):91–96.
Lamey P.J., Hyland P.L.: Changing epidemiology of herpes simplex virus type 1 infections. *Herpes* 1999; 6:20–24.
Lévy-Bruhl D., Bousquet V., King L.A. et al.: The current state of introduction of HPV vaccination into national immunisation schedules in Europe: results of the VENICE 2008 survey. Country specific VENICE gate keepers and contact points. *Eur J Cancer* 2009; 45(15): 2709–2713.
Mammas I.N., Sourvinos G., Spandidos D.A.: Human papilloma virus (HPV) infection in children and adolescents. *Eur J Pediatr* 2009; 168(3):267–273.
Marquart J.D., Trakimas C.A., Sawchuk W.S., et al.: Human papillomavirus 57-induced extensive, recalcitrant cutaneous verrucae. *J Am Acad Dermatol* 2006; 55(5):907–908.
McIver C.J., Jacques C.F., Chow S.S., Munro S.C., Scott G.M., Roberts J.A., Craig M.E., Rawlinson W.D.: Development of multiplex PCRs for detection of common viral pathogens and agents of congenital infections. *J Clin Microbiol* 2005; 43:5102–5110.
Moutschen M., Léonard P., Sokal E.M., Smets F., Haumont M., Mazzu P., Bollen A., Denamur F., Peeters P., Dubin G., Denis M.: Phase I/II studies to evaluate safety and immunogenicity of a recombinant gp350 Epstein-Barr virus vaccine in healthy adults. *Vaccine* 2007; 25(24):4697–4705.
Muller W.J., Jones C.A., Koelle D.M.: Immunobiology of herpes simplex virus and cytomegalovirus infections. *Curr. Immunol. Rev* 2010; 6:38–55.
Murray P.R., Rosenthal K.S., Kobayashi G.S., Pfaller M.A.: *Medical Microbiology*, fourth edition. St. Louis: A Harcourt Health Sciences Company, Missouri 2002.
National Cancer Institute, US National Institutes of Health. *Human papillomaviruses and cancer: questions and answers*. Available at: http://www.cancer.gov/cancertopics/factsheets/risk/HPV. Accessed April 28, 2008.
Reszka E., Jegier B., Wasowicz W., Lelonek M., Banach M., Jaszewski R.: Detection of infectious agents by polymerase chain reaction in human aortic wall. *Cardiovasc Pathol* 2008; 17:297–302.
Ressel G.W.: CDC releases 2002 guidelines for treating STDs. *Am. Fam. Physican* 2002; 66: 1777–1780.
Rice P.S., Mant C., Cason J. et al.: High prevalence of human papillomavirus type 16 infection among children. *J Med Virol* 2000; 61(1):70–75.
Roizman B., Sears A.E.: Herpes simplex viruses and their replication. In: Fields B.N., Knipe D.M., Howley P.M. (eds), *Fields Virology*, 3th ed., Vol. 2. Lippincott-Raven, Philadelphia: 1996:2231–2295.
Schleiss M.R.: Cytomegalovirus Vaccine Development. *Curr Top Microbiol Immunol* 2008; 325:361–382.
Schleiss M.R.: *Cytomegalovirus infection*. Available at: http://emedicine.medscape.com/article/963090-overview (updated Nov 18, 2009).
Shin C.H., Park G.S., Hong K.M., Paik M.K.: Detection and typing of HSV-1, HSV-2, CMV and EBV by quadruplex PCR. *Yonsei Med* J 2003; 44:1001–1007.
Smith E.M., Swarnavel S., Ritchie J.M. et al.: Prevalence of human papillomavirus in the oral cavity/oropharynx in a large population of children and adolescents. *Pediatr Infect Dis J* 2007; 26(9):836–840.
Stanberry L.R., Roenthal S.L.: The epidemiology of herpes simplex virus infections in adolescents. *Herpes* 1999; 6:12–15.

Steben M., Sacks S.: Genital herpes: the epidemiology and control of a common sexually transmitted disease. *Can J Hum Sex* 1997; 6:2.
Summersgill K.F., Smith E.M., Levy B.T., et al.: Human papillomavirus in the oral cavities of children and adolescents. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2001; 91(1): 62–69.
Syrjänen S.: Human papillomavirus infections and oral tumors. *Med Microbiol Immunol 2003*; 192(3):123–128.
Wilson S.S., Fakioglu E., Herold B.C.: Novel approaches in fighting herpes simplex virus infections. *Expert Rev Anti Infect Ther* 2009; 7:559–568.

Joanna Pacholska-Bogalska, Julia Durzyńska,
Anna Goździcka-Józefiak

# Analysis of a polymorphism in the regulatory region of the insulin-like growth factor 1 *(IGF-1)* gene in adolescents with physical developmental disorders and selected diseases

**Abstract:** Insulin-like growth factor 1 is a crucial regulator of growth and development of an organism. This protein is encoded by the gene located on chromosome 12, with transcription regulated by two promoters, referred to as P1 and P2, which are situated before exon 1 and 2 respectively. Main promoter region P1 consists of a DNA fragment situated upstream from the first transcription start site 1630 base pairs long and a fragment of exon 1 – 5'UTR, 322 base pairs long. Promoter sequence polymorphisms are thought to influence *IFG-1* expression level. These polymorphisms also correlate with some diseases in humans, such as cardiovascular disease, type 2 diabetes or various types of cancer.

The present study investigates the sequence of P1 regulatory region of the *IGF-1* gene in a control group of 100 children and a study group of 84 adolescents with physical developmental disorders. In total, single nucleotide polymorphism at –383 C/T site was detected in 11.9% of the group samples of children with physical developmental disorders or other diseases. The polymorphism of cytosine-adenine (CA) repeats, located 1kbupstream from the first transcription start site of *IGF-1*, was also analyzed. It was revealed, that the $(CA)_{19}$. allele was the most common one in the population under study. In the majority of cases the difference in particular allele frequency in the study groups in comparison with the control group had no statistical significance. However, in the group of young people with congenital heart diseases, essential difference regarding the frequency of alleles could be observed, since the frequency of the $(CA)_{19}$ allele was lower and the frequency of the $(CA)_{21}$ allele was higher than in the control group.

**Key words:** insulin-like growth factor 1 (IGF-1), *IGF-1* promoter polymorphism

## Introduction

Apart from the growth hormone (GH), insulin-like growth factor 1 (*IGF-1*) is the most important factor responsible for growth regulation of an organism. It is a small protein of one polypeptide chain, consisting of 70 amino acids with a molecular mass of 7649 Da. This protein and insulin-like growth factor 2 (*IGF-2*) both belong to the same family as insulin and relaxin, demonstrating structural affinity [LeRoith 2001].

IGF-1 is produced by the cells of most tissues, predominantly the liver. It affects target cells through a specialized receptor (IGF-1R), thus promoting growth, differentiation and proliferation of cells, as well as causing an anti-apoptotic effect. IGF-1 acts in target cells in the endocrine, paracrine and autocrine way, and circulating IGF-1 level does not necessarily reflect its real concentration in local tissues [Kaplan and Cohen 2007]. The insulin-like growth factor 1 circulates in the bloodstream in nanomolar concentrations and in the majority of cases binds with one of the six insulin-like growth factor binding proteins (*IGFBP*) that modulate its biological activity [Daughaday 2000; LeRoith 2001].

The significance of IGF-1 for an organism can be seen in the multiple and diverse actions it is engaged in. Many growth hormone actions are mediated by IGF-1 [Kaplan and Cohen 2007], primarily in stimulation of growth and development an organism as early on as embryogenesis [Yakar 1999; Le Roith 2001]. By influencing cell cycle regulation, IGF-1 also promotes differentiation and proliferation of cells in tissues, for instance in bones and muscles, in cells originating from hematopoietic and lymphatic systems or in pancreas β-cells. In addition, IGF-1 exerts anti-apoptotic effect, increasing cell survivability in response to environmental stimuli (ex. hypoxia) and chemical stimuli (ex. chemotherapeutic agents) [LeRoith 2001]. Moreover, this protein also has an acute anabolic influence upon metabolism of proteins and carbohydrates through the increase of cellular uptake of amino acids and glucose, as well as stimulation of protein and glycogen synthesis [LeRoith 2001; Vaessen 2001]. Furthermore, IGF-1 inhibits protein degradation in muscles, modulates immune response through regulation of cytokine production and cell proliferation (clonal expansion) and neutralizes the unfavourable nitrogen equilibrium resulting from malnutrition. IGF-1 promotes steroidogenesis in the reproductive system and adrenal glands, stimulates progesterone production in follicular cells and enhances a synergistic effect on FSH and estradiol [LeRoith 2001]. It may also play a role in longevity regulation in animals and humans [Gems 2001; Carter 2002].

The gene encoding the IGF-1 protein is represented by a single-copy in the human genome. It is located on chromosome 12 (12 q22-24.1) [Rotwein 1986] and covers more than 90 kilo base pairs. The 6 exons and 5 introns can be distinguished in the structural compound of the gene. Study results indicate, that a mature molecule of the IGF-1 protein is encoded by exons 3 and 4 only, while alternatively spliced exons 5 and 6 encode peptide E, present in the precursor molecule of the hormone [Mittanck 1997].

Mammalian *IGF-1* gene transcription is controlled by two promoters, referred to as P1 and P2, situated before exon 1 and 2 respectively [Mittanck 1997; Wang 2000]. Main promoter region P1 consists of the DNA fragment, situated upstream from the transcription start site, 1630 base pairs long and fragment of exon 1, the so-called 5'UTR, 322 base pairs long. That exon 1 5'UTR fragment of exon 1 is indispensable for regular and efficient transcription of this gene and remains its most conserved part [Mittanck 1997].

In the P1 promoter, no sequences typical for promoter regions, such as TATA-box, CCAAT-box or GC-rich sequences have been detected [Mittanck 1997]. This could probably result in multiple transcription initiation sites in the *IFG-1* gene in humans and different animal species [Mittanck 1997; Wang 2000].

In the *IFG-1* gene regulatory sequence, multiple sites for potential specific binding of transcription factors, which could regulate the expression of the gene by either enhancing or inhibiting it have been detected [Yakar 1999, Zhou 1999]. It was established, that some of them were essential not only for *IFG-1* gene expression, but also for its metabolic regulation. It was revealed, that there was a decreased ability of the studied nuclear factors to affect some *IFG-1* gene regions in liver tissues of diabetic rats [Kaytor 2001].

The *IFG-1* expression and secretion are regulated during development of an organism [Shoba 1999]. Low after birth, the *IGF-1* concentration progressively increases during childhood and puberty (10–100 fold) and progressively decreases thereafter. These changes correspond with growth hormone secretion [Daughaday 2000; Le Roith 2001]. Beside the growth hormone, *IGF-1* synthesis and secretion is also affected by such factors as age, gender and diet. Serum *IGF-1* concentration is reduced and resistance to growth hormone is observed in catabolic states like starvation, low calorie and low protein diets, severe injuries and septicemia [Yakar 1999; Daughaday 2000; LeRoith 2001]. Major significance is attributed to the expression of signaling molecules mediating in response to GH [Shoba 1999], local factors [Zhou 1999] and other hormones, for instance insulin [Kaytor 2001], parathyroid hormone and sexual hormones, with estrogens inhibiting IGF-1 secretion and androgens promoting it [Daughaday 2000]. Diseases and other states of an organism are also of great importance. In diabetes, *IGF-1* serum concentration level drops as a result of hypoinsulinemia, while diabetes-induced regulation of synthesis has a tissue-specific character [Zarrilli 1994]. During pregnancy, however, even though GH serum level increases significantly, *IGF-1* serum level rises only slightly because of a high estrogen concentration in pregnant women [Daughaday 2000]. Increased *IGF-1* expression occurs in regenerating tissues, though the effect depends on the damaged tissue type [Zarrilli 1994].

Despite the number of factors that can influence *IGF-1* levels, it has been estimated that up to 60% of the variability has a genetic basis. Recent investigations suggest an association between cytosine-adenine dinucleotide (CA)n repeat polymorphism of *IGF-1* gene promoter region (located 1 kilo base pairs upstream from the first transcription start site) and *IGF-1* levels. The number of CA repeats in the promoter region is inversely correlated with transcription activity [Yazdanpanah 2006a].

Decreased IGF-1 serum concentration is associated with a higher risk of some diseases, such as diabetes [Vaessen 2001] or cardiovascular system diseases [Juul 2002; Laughlin 2004], whereas increased *IGF-1* serum concentration level is associated with a higher risk of breast [Hankinson 1998; Del Giudice 1998], prostate [Chan 1998; Signorello 1999], lung [Yu 1999], colon and rectum cancers [Ma 1999; Palmqvist 2002] and leukaemia in children [Petridou 1999]. Increased *IFG-1* gene expression is also often detected in many cases of thyroid gland cancer [Maiorano 2000].

However, circulating *IGF-1* level does not necessarily reflect its real concentration in local tissues. Genetic polymorphism, that may affect the expression of the *IGF-1* gene, could serve as a better indicator of exposure to *IGF-1*, locally and systemically [Sandhu 2002].

## Objective

The aim of the present study was to analyze selected polymorphisms of the regulatory region of the insulin-like growth factor 1 (*IGF-1*) gene in the genome of children selected from our trial for reasons of their physical developmental disorders or other diseases.

Two fragments of the *IGF-1* promoter region were chosen for the analysis. The first fragment is situated close to the first transcription start site of the *IGF-1* gene, located from – 476 to –148 bp. Multiple transcription factor binding sites are located within this fragment of the gene. Therefore, mutations occurring in these sequences may potentially affect the level of *IGF-1* gene expression and the amount of the protein produced.

However, the second fragment, located from –723 to –591 bp, contains cytosine-adenine (CA)n repeats polymorphism, which is associated with some diseases in humans.

## Material and methods

### Clinical samples

Study material consisted of the DNA isolated from oral and pharyngeal swabs sampled from subjects. Oral squamous cells from all the studied subjects were sampled as swabs and oral rinses for immediate laboratory processing. Buccal cells from swabs were dispersed into bijoux bottles already containing 5 ml of buccal saline solution (oral rinse from the same individual) in order to enrich its content of nucleated squamous cells. Total DNA isolation from oral squamous cells was performed with a ready-to-use set, A&A Biotechnology Swab (cat. nr 025–100, A&A

Biotechnology, Gdynia, Poland). The isolated DNA subsequently served as a template in amplification reaction by PCR.

The study population consisted of 97 boys and 87 girls, aged 10–18. Informed consent was obtained from parents of all pupils taking part in the ADOPOLNOR project.

## Analysis of polymorphism in *IGF-1* gene

Genomic DNA was used for the amplification of the fragment of the P1 *IGF-1* regulatory region, located from –476 to –148 bp, by polimerose chain reaction (PCR) and then analyzed with single stranded conformation polymorphism (SSCP) and sequencing methods, as it was described previously [Obrępalska-Stęplowska 2003].

For the second fragment of *IGF-1* promoter analysis, PCR was performed using primers designed to amplify the polymorphic (CA)n repeat of the human *IGF-1* gene. The reaction was carried out in a final volume of 15 $\mu$l, containing 100 ng genomic DNA, 3.75 pmol forward primer (5'-AAGAAAACACACTCTGGCAC-3') labeled with FAM, 3.75 pmol reverse primer (5'-ACCACTCTGGGAGAAGGG TA-3'), 0.1 mM deoxy-NTP, 1.5 mM $MgCl_2$, 1X PCR buffer and 0.6 U HiFi DNA Polymerase (Novazym, Poland, Cat. no. N1003-05). PCR was performed using thermal cycler (Tgradient Thermocycler, Biometra, Germany) with the parameters: 94°C for 4 min; 28 PCR cycles of 5 sec at 94°C, 30 sec at 60°C and completed with a final extension for 30 min. at 65°C. Analysis of PCR products sizes was performed on an automated sequencing apparatus (ABI 3130xl) and determined in comparison with the internal GS600LIZ size standard (Applied Biosystems). The estimation of CA repeat numbers in each analyzed specimen was based on an extrapolation to previously developed specific alleleic ladder. The ladder marker consisted of 14 sequenced amplicons representing alleles with 7, 9, 11, 13, 23 CA repeats.

## Statistical analysis

Sample-based statistical analysis was performed using the two-sided Student's t test method for fraction comparison. The 5% error in concluding was assumed, and $p<0.05$ indicated statistically significant differences. The analysis was performed using STATISTICA8.0 (StatSoft, Poland) software.

## Computer analysis

The promoter region sequences of the *IGF-1* gene were analyzed using databases and software of AliBaba2.1, Patch 1.0 and P-Match Public (http://www.gene-regulation.com).

## Results

The P1 promoter region of the *IGF-1* gene was analyzed in the DNA isolated from oral and pharyngeal swabs sampled from 184 students taking part in the ADOPOLNOR project (www.adopolnor.eu). The control group consisted of 100 pupils without developmental disorders or other diseases. The rest of the participants were selected on the basis of qualifying for one of the prophylactic groups: children with cardiovascular system diseases (group 8) – 17 cases, children with congenital heart diseases (group 8a) – 12 cases, children with urinary system chronic diseases and malformations (group 9) – 7 cases, children with diabetes (group 13) – 5 cases, children with thyroid gland diseases (group 14) – 7 cases and children with physical developmental disorders (group 4a) – 36 cases. Detailed characteristics reported in Table 1 also include gender, age, height and body mass index (BMI) of adolescents in the study groups.

The isolated DNA served as a template in amplification reaction and subsequent analysis of the selected fragments of the *IGF-I* gene promoter.

The fragment of the P1 promoter, located from –476 to –148 bp in the DNA sequence, was analyzed by PCR-SSCP and sequencing methods. As a result of the conducted study, no nucleotide sequence variations were found in the control group, nor were they revealed in the group of children with thyroid gland diseases (prophylactic group 14) or in the group of adolescents with physical developmental disorders (prophylactic group 4a).

However, single nucleotide polymorphism (SNP) at site –383 C/T of the regulatory region was found in 17,65% (3 out of 17 cases) of adolescents with cardiovascular system diseases (prophylactic group 8), in 25% (3 out of 12 cases) of adolescents with congenital heart diseases, in 28,57% (2 out of 7 cases) of adolescents with chronic urinary system diseases and malformations and in 40% (2 out of 5 cases) children with diabetes. The difference of SNP frequency at site –383 C/T of the *IGF-1* gene promoter in the control and study groups were statistically significant ($p<0.001$).

The computer analysis of how sequence variation at site –383 C/T of *IGF-1* gene promoter impacts the probability of transcription factor binding was done with program AliBaba 2.1, which uses TRANSFAC database (http://www.gene-regulation.com). This analysis revealed, that variation at this site eliminates potential binding site for transcription factor Sp1.

We also analyzed the polymorphism of CA repeats situated in the P1 promoter region located 1kb upstream from the first transcription start site of the *IGF-1* gene. The CA repeat sequences in the *IGF-1* promoter among the study group ranged from 11 to 23, although 19 repeats (allele ($CA_{19}$))were most common, with an allele frequency of 68,5% in the control group. The frequency of particular alleles is reported in Table 2. In the majority of cases the difference in particular allele frequency in the study groups in comparison with the control group had no statistical significance. The only substantial difference in $(CA)_{19}$ and $(CA)_{21}$ allele frequency was observed in the group of children with congenital

heart diseases. This group demonstrated allele $(CA)_{19}$ frequency of 41.67% against 68,5% in the control group and allele $(CA)_{21}$ frequency of 33.33% against 5.88% in the control group.

Table 1. Characteristics of the examined groups of children

| Group | Age | Boys n | Boys Height Mean | Boys Height SD | Boys BMI Mean | Boys BMI SD | Girls n | Girls Height Mean | Girls Height SD | Girls BMI Mean | Girls BMI SD | total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Control group | 10–12 | 13 | 144.09 | 7.42 | 18.91 | 4.30 | 14 | 147.89 | 7.23 | 19.32 | 2.78 | |
| | 13–15 | 24 | 166.36 | 9.10 | 20.29 | 3.76 | 25 | 161.00 | 7.43 | 18.47 | 2.35 | |
| | 16–18 | 15 | 180.73 | 5.70 | 21.32 | 2.14 | 9 | 162.64 | 6.38 | 20.59 | 1.91 | |
| | total | 52 | | | | | 48 | | | | | 100 |
| Adolescents with cardio-vascular diseases | 10–12 | 4 | 155.81 | 14.14 | 23.07 | 5.61 | 0 | | | | | |
| | 13–15 | 4 | 168.31 | 10.78 | 24.55 | 5.95 | 1 | 165.55 | 0.00 | 18.25 | 0.00 | |
| | 16–18 | 6 | 178.31 | 8.04 | 24.15 | 9.29 | 2 | 162.35 | 1.63 | 30.20 | 18.50 | |
| | total | 14 | | | | | 3 | | | | | 17 |
| Adolescents with congenital heart defects | 10–12 | 0 | | | | | 2 | 149.98 | 1.94 | 17.86 | 1.06 | |
| | 13–15 | 5 | 157.61 | 5.47 | 20.00 | 2.95 | 1 | 173.30 | 0.00 | 20.72 | 0.00 | |
| | 16–18 | 0 | | | | | 4 | 161.88 | 7.62 | 20.90 | 1.38 | |
| | total | 5 | | | | | 7 | | | | | 12 |
| Adolescents with chronic diseases and defects of urinary system | 10–12 | 1 | 142.05 | 0.00 | 21.33 | 0.00 | 0 | | | | | |
| | 13–15 | 0 | | | | | 1 | 152.60 | 0.00 | 22.17 | 0.00 | |
| | 16–18 | 2 | 175.23 | 2.09 | 20.01 | 1.05 | 3 | 166.98 | 2.24 | 24.44 | 2.77 | |
| | total | 3 | | | | | 4 | | | | | 7 |
| Diabetic adolescents | 10–12 | 1 | 171.25 | 0.00 | 17.07 | 0.00 | 0 | | | | | |
| | 13–15 | 1 | 146.75 | 0.00 | 15.50 | 0.00 | 2 | 164.83 | 9.09 | 21.49 | 5.41 | |
| | 16–18 | 1 | 186.35 | 0.00 | 20.66 | 0.00 | 0 | | | | | |
| | total | 3 | | | | | 2 | | | | | 5 |
| Adolescents with thyroid diseases | 10–12 | 0 | | | | | 0 | | | | | |
| | 13–15 | 1 | 180.05 | 0.00 | 20.68 | 0.00 | 2 | 159.88 | 11.00 | 21.34 | 2.87 | |
| | 16–18 | 1 | 166.60 | 0.00 | 22.01 | 0.00 | 3 | 160.95 | 10.17 | 23.03 | 3.93 | |
| | total | 2 | | | | | 5 | | | | | 7 |
| Adolescents with physical development disorders | 10–12 | 4 | 155.41 | 7.04 | 18.98 | 4.75 | 6 | 150.23 | 8.61 | 24.23 | 5.72 | |
| | 13–15 | 8 | 165.18 | 8.30 | 19.97 | 4.64 | 7 | 158.03 | 10.16 | 21.82 | 4.62 | |
| | 16–18 | 6 | 181.50 | 8.83 | 29.11 | 6.03 | 5 | 161.63 | 9.23 | 26.03 | 7.46 | |
| | total | 18 | | | | | 18 | | | | | 36 |
| Total | | 97 | | | | | 87 | | | | | |
| | | | | | | | | | | | Total | 184 |

Table 2. Allele distribution of the IGF-1 promoter polymorphism

| The repeated CA units | Control group | Adolescents with cardio-vascular diseases | Adolescents with congenital heart defects | Adolescents with chronic diseases and defects of urinary system | Diabetic adolescents | Adolescents with thyroid diseases | Adolescents with physical development disorders |
|---|---|---|---|---|---|---|---|
| 11 | 1.50% | – | – | – | 10% (p=0.0564) | – | 1.39% (p=0.9470) |
| 17 | 1.50% | – | – | – | 10.00% (p=0.0564) | – | 1.39% (p=0.9470) |
| 18 | 6.50% | – | – | – | – | 14.29% (p=0.2710) | 5.56% (p=0.7778) |
| 19 | 68.50% | 79.41% (p=0.2003) | 41.67% (p=0.0095) | 71.43% (p=0.8194) | 50.00% (p=0.2238) | 50.00% (p=0.1552) | 66.67% (p=0.7755) |
| 20 | 15.00% | 11.76% (p=0.6206) | 20.83% (p=0.4581) | 14.29% (p=0.5427) | 20.00% (p=0.6681) | 35.71% (p=0.0441) | 15.28% (p=0.9546) |
| 21 | 5.50% | 5.88% (p=0.9288) | 33.33% (p=0.0000) | 14.29% (p=0.1846) | 10.00% (p=0.5503) | – | 8.33% (p=0.1465) |
| 22 | 1.00% | 2.94% (p=0.3535) | – | – | – | – | 1.39% (p=0.7861) |
| 23 | 0.50% | – | – | – | – | – | – |

## Discussion

Polymorphisms in the *IGF-I* gene regulatory sequence are thought to affect serous IGF-1 protein level and organism growth variability, including birth size, height and weight of humans [Vaessen 2002]. They are also associated with some diseases, such as breast [Yu 2001, Missmer 2002; De Lellis 2003, Wen 2005], prostate [Tsuchiya 2005, Schildkraut 2005], colon and rectum cancers [Wong 2005] as well as cardiovascular disease and type 2 diabetes [Vaessen 2001; Yazdanpanah 2006a].

One of the most thoroughly researched *IGF-1* polymorphisms is the sequence of multiple cytosine-adenine dinucleotide repeats (CA repeats), situated in the 5' regulatory region of the *IGF-1* gene about 1 kilo base pairs upstream from the first transcription start site [Rotwein 1986]. The number of CA repeats ranges between 10 and 24, with the most common allele containing 19 CA repeats in the Caucasian population [Rosen 1998; Voorhoeve 2006].

In our study, allele containing 19 CA repeats was also found to be the most common, with an allele frequency of 68.5% in the control group. In study groups, except for the group of children with congenital heart diseases, this allele frequency ranged from 50% to 79,49%, while difference in the study groups in comparison with the control group had no statistical significance. Lower frequency of allele $(CA)_{19}$ in children with diabetes or with thyroid gland diseases (50%) might

result from the small number of participants in these groups. The obtained allele frequency results concurred with those of Vaessen et al. [2001].

The most interesting result was obtained in the group of children with congenital heart diseases (prophylactic group 8a), where allele $(CA)_{19}$ frequency of 41.67% was significantly lower, than in the control group, whereas allele $(CA)_{21}$ frequency of 33.33% was higher, than in the control group.

IGF-1 is an important factor that affects cardiac development via increasing DNA and protein synthesis, reduction of protein degradation and participation in proliferation and maturation of cardiomyocytes during the early stages of the human development. It also improves cardiac contractility, cardiac output, stroke volume and injection fraction. Moreover, IGF-1 participates in glucose metabolism, lowers insulin level coupled with enhancing cell sensitivity to it, improves lipid profile, which significantly impacts heart and blood vessels functioning. In addition, IGF-1 improves cardiac function after myocardial infarction through stimulation of contractility and promotion of tissue remodelling. This protein may also influence heart work through prevention of apoptosis, which contributes to heart failure [Ren 1999].

The study of other genes suggest that polymorphic (CA)n repeats in their promoter regions affect their transcription activity [Tae 1994] and that the number of CA repeats is inversely correlated with their transactivation [Gebhardt 1999]. The way in which the number of CA repeats in the *IGF-1* regulatory region affects the level of mRNA *in vitro* and *in vivo* transcriptions remains unknown. Studies of association between *IGF-1* polymorphism and *IGF-1* level in blood serum have been inconsistent. While some of them reveal, that *IGF-1* level in blood serum increased with the number of alleles containing 19 CA repeats (homozygosity of 19 CA repeats was associated with higher *IGF-1* level) [Vaessen 2001; Missmer 2002], other studies indicate, that such homozygotes had a lower *IGF-1* level in blood serum, than all other genotypes [Rosen 1998; Jernstrom 2001], whereas subsequent studies show no connection between polymorphism and circulating *IGF-1* level [Allen 2002; Frayling 2002; Kato 2003; Wen 2005]. Nevertheless, studies conducted in a large ethnically homogenous Caucasian population indicate, that there is a link between IGF-1 level in blood serum and polymorphism of number of CA repeats. These studies revealed, that circulating IGF-1 level was the highest among subjects with alleles $(CA)_{19}$ or $(CA)_{20}$, whereas subjects with alleles shorter than 19 CA repeats or longer than 20 CA repeats had a lower serous IGF-1 level [Rietveld 2004; Bleumink 2004]. Conclusions were made, that absence of the $(CA)_{19}$ allele was closely related not only to a lower IGF-1 level in blood serum, but also to a weaker growth [Vaessen 2001].

There is evidence, that IGF-1 plays a significant role in cardiovascular pathophysiology and heart failure. Low IGF-1 level in blood serum is associated with a higher risk of cardiovascular disease, such as ischemic heart disease [Juul 2002; Sandhu 2002; Laughlin 2004]. It was also revealed, that subject aged 55–75 lacking $(CA)_{19}$ and/or $(CA)_{20}$ alleles manifested a lower circulating IGF-1 level and a higher risk of incident heart failure [Vasen 2003, Bleumink 2004]. Moreover, non-carriers of $(CA)_{19}$ allele showed a higher risk of type 2 diabetes or myocardial infarction

risk. Particularly in the case of diabetics, the relative risk of myocardial infarction was particularly high in subjects lacking $(CA)_{19}$ allele [Vaessen 2001]. At the same time, subjects with $(CA)_{19}$ and/or $(CA)_{20}$ alleles had a higher risk of death during myocardial infarction. Thus, the more frequent $(CA)_{19}$ and/or $(CA)_{20}$ alleles were in the genome, the higher risk of death rate was. However, no association between survival and polymorphysm of the *IGF-1* gene promoter was established in the whole population under study [Yazdanpanah 2006b].

For reasons of its invasive character and costs, the analysis of serous IGF-1 level wasn't conducted in the ADOPOLNOR project. Moreover, the number of subjects in study groups would have hampered in establishing reliable correlations between CA repeats polymorphism of the *IGF-1* and circulating IGF-1 level.

In the second analyzed fragment of the *IGF-1* promoter region, we identified single nucleotide polymorphism at site –383 C/T, which was detected in total in 11,9% of the group samples of children with physical developmental disorder or selected diseases.

The performed computer analysis revealed, that variation at –383 C/T site of *IGF-1* gene promoter eliminates potential binding site for transcription factor Sp1, which as a result might influence variability in *IGF-1* gene expression.

Ubiquitous in mammalian cells, Sp1 belongs to the family of nuclear proteins, which modulate genes transcription. Single or multiple binding sites of Sp1 (the so-called GC/GT-boxes) have been identified in promoters and gene-enhanancing sequences of many genes engaged in almost all cell processes, including housekeeping, tissue-specific and viral genes [Suske 1999; Li 2004]. Sp1 protein shares structural homology with Sp3 protein, which has affinity for binding Sp1 sites [Li 2004]. Sp1 and Sp3 can both act as negative or positive regulators of gene expression [Suske 1999; Li 2004], depending on the promoter and cell background [Yu 2003; Li 2004]. Sp1 acts directly with TAF (TATA-binding protein associated factor) and other nuclear proteins [Safe and Abdelrahim 2005]. Sp1-dependent transactivation is also conditioned by multiprotein nuclear complexes, referred to as CRSP (cofactors required for Sp1 coactivation). Interactions between Sp1 and other proteins may affect in various ways the Sp1-dependent transactivation and thus may modulate gene expression [Safe and Abdelrahim 2005].

In summary, the results from our study suggest association between polymorphism of the *IGF-1* gene promoter and susceptibility to heart diseases, but extended analysis of larger study group are needed.

## References

Allen N.E., Davey G.K., Key T.J., Zhang S., Narod S.A.: Serum insulin-like growth factor I (GF-1) concentration in men is not associated with the cytosine-adenosine repeat polymorphism of the GF-1 gene. *Cancer Epidemiol Biomarkers Prev* 2002; 11:319–320.

Bleumink G.S., Rietveld I., Janssen J.A., van Rossum E.F., Deckers J.W., Hofman A., Witteman J.C., van Duijn C.M., Stricker B.H.: Insulin-like growth factor-I gene polymorphism and risk of heart failure (the Rotterdam Study). *Am J. Cardiol* 2004; 94:384–386.

Carter C.S., Ramsey M.M., Sonntag W.E.: A critical analysis of the role of growth hormone and IGF-I in aging and lifespan. *Trends Genet* 2002; 18:259–301.

Chan J.M., Stampfer M.J., Giovannucci E., Gann P.H., Ma J., Wilkinson P., Hennekens C.H., Pollak M.: Plasma insulin-like growth factor 1 and prostate cancer risk: a prospective study. *Science* 1998; 279:563–565.

Daughaday W.H.: Growth hormone axis overview – somatomedin hypothesis. *Pediatr Nephrol* 2000; 14:537–540.

De Lellis K., Ingles S., Kolonel L., McKean-Cowdin R., Henderson B., Stanczyk F., Probst-Hensch N.M.: GF-1 genotype, mean plasma level and breast cancer risk in the Hawaii/Los Angeles multiethnic cohgort. *Br J Cancer* 2003; 88:277–282.

Del Giudice M.E., Fantus I.G., Ezzat S., McKeown-Eyssen G., Page D., Goodwin P.J.: Insulin and related factors in premenopausal breast cancer risk. *Breast Cancer Res Treat* 1998; 47: 111–120.

Frayling T.M., Hattersley A.T., McCarthy A., Holly J., Mitchell S.M., Gloyn A.L., Owen K., Davies D., Smith G.D., Ben-Shlomo Y.: A putative functional polymorphism in the IGF-I gene: association studies with type 2 diabetes, adult height glucose tolerance and fetal growth in U.K. populations. *Diabetes* 2002; 51:2313–2316.

Gebhardt F., Zanker K.S., Brandt B.: Modulation of epidermal growth factor receptor gene transcription by a polymorphic dinucleotide repeat in intron 1. *J Biol Chem* 1999; 274: 13176–13180.

Gems D., Partridge L.: Insulin/IGF signaling and aging: seeing the bigger picture. *Curr Opin Genet Dev* 2001; 11:287–292.

Hankinson S.E., Willett W.C., Colditz G.A., Hunter D.J., Michaud D.S., Deroo B., Rosner B., Speizer F.E., Pollak M.: Circulating concentrations of insulin-like growth factor–1 and risk of breast cancer. *Lancet* 1998; 351:1393–1396.

Jernstrom H., Deal C., Wilkin F., Chu W., Tao Y., Majeed N., Hudson T., Narod S.A., Pollak M.: Genetic and non-genetic factors associated with variation of plasma levels of insulin-like growth factor-I and insulin –like growth factor binding protein–3 in healthy premenopausal women. *Cancer Epidemiol Biomarkers Prev* 2001; 10:377–384.

Juul A., Scheike T., Davidsen M., Gyllenborg J., Jorgensen T.: Low serum insulin-like growth factor I is associated with increased risk of ischemic heart disease: a population-based case–control study. *Circulation* 2002; 106:939–944.

Kaplan S.A., Cohen P.: Review: The somatomedin hypothesis 2007: 50 Years Later. *J Clin Endocrinol Met* 2007; 92:4529–4535.

Kato I., Eastham J., Li B., Smith M., Yu H.: Genotype-phenotype analysis for the polymorphic CA repeat in the insulin-like growth factor-I (IGF-I) gene. *Eur J Epidemiol* 2003; 18: 203–209.

Kaytor E.N., Zhu J.L., Pao C-I., Phillips L.S.: Physiological concentrations of insulin promote binding of nuclear proteins to the insulin-like growth factor I gene. *Endocrinology* 2001; 142:1041–1046.

Laughlin G.A., Barrett-Connor E., Criqui M.H., Kritz-Silverstein D.: The prospective association of serum insulin-like growth factor I(IGF-I) and IGF-binding protein–1 levels with all cause and cardiovascular disease mortality in older adults: the Rancho Bernardo Study. *J Clin Endocrinol Met* 2004; 89:114–120.

LeRoith D., Bondy C., Yakar S., Liu J-L., Butler A.: The somatomedin hypothesis: 2001. *Endocr Rev* 2001; 22:53–74.

Levy D., Wilson P.W.: Serum insulin-like growth factor I and risk for heart failure in elderly individuals without a previous myocardial infarction: the Framingham Heart Study. *Ann Intern Med* 2003; 139:642–648.

Li L., Shihua H., Jian-Min S., Davie J.: Gene regulation by Sp1 and Sp3. *Biochem Cell Biol* 2004; 82:460–471.

Ma J., Pollak M.N., Giovannucci E., Chan J.M., Tao Y., Hennekens C.H., Stampfer M.J.: Prospective study of colorectal cancer risk in men and plasma levels of insulin-like growth factor (IGF)-I and IGF-binding protein–3. *J Natl Cancer Inst* 1999; 91:620–625.

Maiorano E., Ciampolillo A., Viale G., Maisonneuve P., Ambrosi A., Triggiani V., Marra E., Perlino E.: Insulin-like growth factor 1 expression in thyroid tumors. *Appl Immunohistochem Mol Morphol* 2000; 8:110–119.

Missmer S.A., Haiman C.A., Hunter D.J., Willett W.C., Colditz G.A., Speizer F.E., Pollak M.N., Hankinson S.E.: A sequence repeat in the insulin-like growth factor–1 gene and risk of breast cancer. *Int J Cancer* 2002; 100:332–336.

Mittanck D.W., Kim S-W., Rotwein P.: Essential promoter elements are located within the 5' untranslated region of human insulin-like growth factor – I exon I. *Mol Cell Endocrinol* 1997; 126:153–163.

Obrępalska-Stęplowska A., Kędzia A., Trojan J., Goździcka-Józefiak A.: Analysis of coding region and promoter sequences of coding and promoter sequences of the IGF-I gene in children with growth disorders presenting with normal and normal level of growth hormone. *J Pediatr Endocrinol Metab* 2003; 16:1267–1275.

Palmqvist R., Hallmans G., Rinaldi S., Biessy C., Stebling R., Ribioli E., Kaaks R.: Plasma insulin-like growth factor 1, insulin-like growth factor binding protein 3 and risk of colorectal cancer: a prospective study in northern Sweden. *Gut* 2002; 50:642–646.

Petridou E., Dessypris N., Spanos E., Mantzoros C., Skalkidou A., Kalmanti M., Koliouskas D., Kosmidis H., Panagiotou J.P., Piperopoulou F., Tzortzatou F., Trichpoulos D.: Insulin-like growth factor-I and binding protein–3 in relation to childhood leukaemia. *Int J Cancer* 1999; 80:494–496.

Ren J., Samson W.S, Sowers J.R.: Insulin-like growth factor I as a cardiac hormone: physiological and pathophysiological implications in heart disease. *J Mol Cell Cardiol* 1999; 31: 2049–2061.

Rietveld I., Janssen J.A., van Rossum E.F., Houwing-Duisternaat J.J., Rivadeneira F., Hofman A., Pols H.A., van Duijn C.M., Lamberts S.W.: A polymorphic CA repeat in the IGF-I gene is associated with gender-specific differences in body height. *Clin Endocrinol* (*Oxf*) 2004; 61: 195–203.

Rosen C.J., Kurland E.S., Vereauldt D., Adler R.A., Rackoff P.J., Craig W.Y., Witte S., Rogers J., Bilezikian J.P.: Association between serum insulin-like growth factor-I (IGF-I) and a simple sequence repeat in *IGF-I* gene: implications for genetic studies of bone mineral density. *J Clin Endocrinol Metab* 1998; 83:2286–2290.

Rotwein P., Pollock K.M., Didier D.K., Krivi G.G.: Organization and sequence of the human insulin-like growth factor I gene. Alternative RNA processing produces two insulin-like growth factor I precursor peptides. *J Biol Chem* 1986; 261:4828–4832.

Safe S., Abdelrahim M.: Sp transcription factor family and its role in cancer. *Eur J Cancer* 2005; 41:2438–2448.

Sandhu M.S., Heald A.H., Gibson J.M., Cruickshank J.K., Dunger D.B., Wareham N.J.: Circulating concentrations of insulin-like growth factor-I and development of glucose intolerance: a prospective observational study. *Lancet* 2002; 359:1740–1745.

Schildkraut J.M., Denmark-Wahnefried W., Wenham R.M., Grubber J., Jeffreys A.S., Grambow S.C., Marks J.R., Moorman P.G., Hoyo C., Ali S., Walther P.J.: *IGF1* $(CA)_{19}$ repeat and IGFBP3 –202 A/C polymorphism and the risk of prostate cancer in black and white men. *Cancer Epidemiol Biomarkers* Prev 2005; 14:403–408.

Shoba L., An M.R., Frank S.J., Lowe Jr. W.L.: Developmental regulation of insulin-like growth factor –I and growth hormone receptor gene expression. *Mol Cell Endocrinol* 1999; 152: 125–136.

Signorello L.B., Brismar K., Bergstrom R., Andersson S.O., Wolk A., Trichopoulos D., Adami H.O.: Insulin-like growth factor-binding protein–1 and prostate cancer. *J Natl Cancer Inst* 1999; 91:1965–1967.

Suske G.: The Sp-family of transcription factors. *Gene* 1999; 238:291–300.

Tae H.J., Luo X., Kim K.H.: Roles of CCAAT/enhancer-binding protein and its binding site on repression and derepression of acetyl-CoA carboxylase gene. *J Biol Chem* 1994; 269: 10475–10484.

Tsuchiya N., Wang L., Horikawa Y., Inoue T., Kakinuma H., Matsuura S., Sato K., Ogawa O., Kato T., Habuchi T.: CA repeat polymorphism in the insulin-like growth factor – I gene is associated with increased risk of prostate cancer and benign prostatic hyperplasia. *Int J Oncol* 2005; 26:225–231.

Vaessen, N., Heutink, P., Janssen, J.A., Witteman, J.C.M., Testers, L., Hofman, A., Lamberts, S.W.J., Oostra, B.A., Pols, H.A.P., van Duijn, C.M.: A polymorphism in the gene for IGF-I. Functional properties and risk for type 2 diabetes and myocardial infarction. *Diabetes*, 2001; 50:637–642.

Vaessen N., Janssen J.A., Heutink P., Hofman A., Lamberts S.W.J., Oostra B.A., Pols H.A.P., van Duijn C.M.: Association between genetic variation in the gene for insulin-like growth factor-I and low birthweight. *Lancet* 2002; 359:1036–1037.

Vasan R.S., Sullivan L.M., D'Agostino R.B., Roubenoff R., Harris T., Sawyer D.B., Voorhoeve P.G., van Rossum E.F., te Velde S.J.: Assotiation between an IGF-I gene polymorphism and body fatness: differences between generations. *Eur J Endocrinol* 2006; 154: 379–388.

Wang L., Wang X., Adamo M.L.: Two putative GATA motifs in the proximal exon 1 promoter of rat insulin-like growth factor I gene regulate basal promoter activity. *Endocrinology* 2000; 141:1118–1126.

Wen W., Gao Y-T., Shu X-O., Yu H., Cai Q., Smith J.R., Zheng W.: Insulin-like growth factor-I gene polymorphism and breast cancer risk in Chinese women. *Int J Cancer* 2005; 13: 307–311.

Wong H-L., DeLellis K., Probst-Hensch N., Koh W-P., van dan Berg D., Lee H-P., Yu M.C., Ingles S.A.: A new single nucleotide polymorphism in the insulin-like growth factor I regulatory region associates with colorectal cancer risk in Singapore Chinese. *Cancer Epidemiol Biomarkers Prev* 2005; 14:144–151.

Yakar, S., Liu J-L., Stannard B., Butler A., Accili D., Sauer B., LeRoith D.: Normal growth and development in the absence of hepatic insulin-like growth factor I. *Proc Natl Acad Sci USA* 1999; 96:7324–7329.

Yazdanpanah M., Sayed-Tabatabaei F.A., Janssen J.A., Rietveld I., Hofman A., Stijnen T., Pols H.A., Lamberts S.W., Witteman J.C., van Duijn C.M.: IGF-I gene promoter polymorphism is a predictor of survival after myocardial infarction in patients with type 2 diabetes. *Eur J Endocrinol* 2006a; 155:751–756.

Yazdanpanah M., Rietveld I., Janssen J.A., Njajou O.T., Hofman A., Stijnen T., Pols H.A., Lamberts S.W., Witteman J.C., van Duijn C.M.: An insulin-like growth factor-I promoter polymorphism is associated with increased mortality in subjects with myocardial infarction in an elderly Caucasian population. *Am J Cardiol* 2006b; 79:1274–1276.

Yu B., Datta P.K., Bagchi S.: Stability of the Sp3-DNA complex is promoter-specific: Sp3 efficiently competes with Sp for binding to promoters containing multiple Sp-sites. *Nucleic Acids Res* 2003; 31:5368–5376.

Yu H., Spitz M.R., Mistry J., Gu J., Hong W.K., Wu X.: Plasma levels of insulin-like growth factor-I and lung cancer risk: a case–control study. *J Natl Cancer Inst* 1999; 9:151–156.

Yu H., Li B.D.L., Smith M., Shi R., Berkel H., Kato I.: Polymorphic CA repeats in the *IGF-I* gene and breast cancer. *Breast Cancer Res Treat* 2001; 70:117–122.

Zarrilli R., Bruni C.B., Riccio A.: Multiple levels of control of insulin-like growth factor gene expression. *Mol Cell Endocrinol* 1994; 101:R1–R14.
Zhou J-L., Pao C-I., Hunter E. Jr., Lin K-W.M., Wu G., Phillips L.S.: Identification of core sequences involved in metabolism-dependent nuclear protein binding to the rat insulin-like growth factor I gene. *Endocrinology* 1999; 140:4761–4771.

# Index